UNIVERSITY OF ALBERTA

THE EFFECT OF HYPEROXIA ON MAXIMAL AND SUBMAXIMAL EXERCISE WITH FIREFIGHTING GEAR AND SELF-CONTAINED BREATHING APPARATUS (SCBA)

by

NEIL DEREK EVES ©

A Thesis submitted to the faculty of graduate studies and research in partial fulfilment of the requirements for the degree of Master of Science.

FACULTY OF PHYSICAL EDUCATION AND RECREATION

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled, The Effect of Hyperoxia on Maximal and Submaximal Exercise Performance with Firefighting Gear and Self Contained Breathing Apparatus, submitted by Neil D. Eves in partial fulfillment of the requirements for the degree of Master of Science.

Dr. S.R. Petersen, Supervisor

Dr. R.L. Jones

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Date 30th SEPTEMBER 1999

DEDICATION

To Mum and Dad

ABSTRACT

To study the effects of hyperoxia on maximal and submaximal (SUB) exercise with a self-contained breathing apparatus (SCBA), 25 adult males completed 3 graded exercise tests (GXT) for maximal oxygen uptake ($\dot{V}O_{2max}$) and 3 SUB trials of 20 min treadmill walking at ventilatory threshold. All exercise tests were completed on separate days, wearing full firefighting gear and SCBA. Two GXTs and two SUB trials used 20.9% O_2 (NOX), while the remaining GXT and SUB used 40% O_2 (HOX). With HOX, $\dot{V}O_{2max}$ and maximal power output (PO_{max}) were increased ($p<0.05$) by 10.1 and 9.3 % respectively. Ventilation ($\dot{V}_E$) and mask pressure (P_{mask}) were significantly lower at normoxic $\dot{V}O_{2max}$ intensity, indicating a decrease in the work of breathing. During the HOX SUB, $\dot{V}_E$ and P_{mask} were also reduced ($p<0.05$) and total gas consumption was 10.8% lower ($p<0.05$). These findings have significant practical implications for occupational safety.

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CHAPTER 1

INTRODUCTION

1.1 Overview of the problem

Hyperoxia (HOX) is the term used to describe an increase in the fraction of oxygen in the inspired air (F_IO_2). For some 75 years, physiologists have known that HOX can improve work tolerance (Hill et al. 1924; Bannister and Cunningham, 1954; Wilson et al. 1975; Byrnes and Mullin, 1981; Plet et al. 1992; Peltonen et al. 1995; Knight et al. 1996). Research has consistently shown that running (Bannister and Cunningham, 1954; Wilson and Welch, 1975), cycling (Adams and Welch, 1980; Plet et al. 1992; Knight et al. 1993) or rowing performance (Peltonen et al. 1995; Nielsen et al. 1998) can be improved at both maximal intensities and during endurance events by increasing the F_IO_2. Obviously, within athletics the use of HOX is unrealistic, simply from a delivery perspective. However, within firefighting where the wearing of a self-contained breathing apparatus (SCBA) is necessary, the administration of HOX would be relatively simple and the benefits could be substantial.

Firefighting is commonly acknowledged as a dangerous and extremely strenuous profession that requires the physical ability to perform for long periods of time in often extremely hostile environments. It is therefore of particular significance to investigate methods that could reduce any of the physiological stresses involved and could increase the safety of the occupation. Such strategies may be immensely beneficial even if the effect was relatively small.

The mechanisms behind the improved performance with HOX are complex and not fully understood. Hill et al. (1924) suggested that any increase in maximal oxygen consumption ($\dot{V}O_{2max}$), must be related to the Fick principle (cardiac output ($\dot{Q}$) x arterial-venous oxygen difference (a-vDO_2)). Unfortunately, few studies have measured $\dot{Q}$, those which have (Ekblom et al. 1975; Horstman et al. 1976) found no significant increases at maximal exercise. It therefore seems more likely that the increases in $\dot{V}O_{2max}$ are related to an increased extraction of O_2 at the muscular level (Ekblom et al. 1975; Horstman et al. 1976; Knight et al. 1993). However, there are a number of very contentious issues that not only surround the magnitude of these increases but also the methods used to analyse the expired gases. Some research has suggested that the increase in $\dot{V}O_{2max}$ can be as high as 22% under hyperoxic conditions (Weltman et al. 1978). Increases of this magnitude have been criticized (Welch and Pedersen 1981; Welch 1982) and appear to be the result of the methods used for gas analysis rather than true values.

Welch and Pedersen (1981) reported that using Douglas bags for expired gas collection tended to overestimate oxygen consumption ($\dot{V}O_2$) with HOX. They attributed this finding to an unavoidable room air contamination of the collected expired sample. However, the authors concluded that this problem appears to be resolved by using a mixing chamber for the expired gas collection, as no significant difference was found using three different calculations for the determination of $\dot{V}O_2$ ((i) $\dot{V}O_2 = \dot{V}_E(F_IO_2 . F_EN_2/F_IN_2 - F_EO_2)$, (ii) $\dot{V}O_2 = \dot{V}_IF_IO_2 - \dot{V}_E F_IO_2$ and iii) $\dot{V}O_{2max} = \dot{V}_I - \dot{V}_E - \dot{V}CO_2$, where F_IO_2 and F_EO_2, are fractional inspired and expired O_2 content respectively and F_IN_2 and F_EN_2 are fractional inspired and expired N_2 respectively).

Welch (1982) postulated, that although increases of approximately10% have been shown (Ekblom et al. 1975; Welch et al. 1977) in the content of arterial oxygen (C_aO_2) with HOX, the content of venous oxygen (C_vO_2) is also elevated, indicating that a-vDO_2 does not increase to the same extent as the rise in O_2 delivery ($\dot{Q} \times C_aO_2$). Welch, therefore suggested that increases in $\dot{V}O_{2max}$ should not exceed 6%, as studies that have measured a-vDO_2 (Stanek et al. 1979 and Welch et al. 1977) with 60-100% O_2, have not exceeded this value. However, only one study with humans (Welch et al. 1977) was used when generating this hypothesis and the validity of their C_aO_2 and C_vO_2 measurements could be influenced by the very small sample size (n=2) and the protocol used for eliciting $\dot{V}O_{2max}$. More recently (Knight et al. 1993) demonstrated a 12% ($p<0.05$) increase in C_aO_2 increased O_2 delivery by 10.9%. This resulted in a (non significant) rise in P_VO_2 and leg $\dot{V}O_{2max}$ increased 8.1%. This finding is in agreement with a number of recent studies that have reported increases in $\dot{V}O_{2max}$ ranging from 11-13%.(Byrnes et al. 1984; Peltonen et al. 1995; Nielsen et al. 1998).

During exercise at submaximal levels with HOX, it has been shown that there is often an increase in $\dot{V}O_2$ (Hughes et al. 1968; Linnarsson et al. 1974; Wilson et al. 1975), while carbon dioxide produced ($\dot{V}CO_2$) remains constant or possibly decreases (Welch et al. 1974; Adams et al. 1986). These findings, and the decrease in the respiratory exchange ratio (RER) (Hughes et al. 1968; Welch et al. 1974; Wilson et al. 1975; Byrnes and Mullin, 1981; Adams et al. 1986) that accompanies them, have led some researchers to the hypothesis that HOX may cause a shift in substrate utilization away from carbohydrate metabolism to an increased utilization of fat (Welch and Pedersen, 1981; Adams et al. 1986). Therefore, HOX may help to maintain aerobic metabolism at higher work intensities, evidenced by the observations of decreased blood lactate levels (Miller et al. 1952; Hughes et al. 1968; Ekblom et al. 1975; Adams and Welch, 1980) and hydrogen ion concentration within the working muscle (Adams and Welch, 1980). This hypothesis may explain the increases in time to exhaustion found in a number of studies (Wilson and Welch, 1975; Adams and Welch, 1980; Plet et al. 1992), as a substrate shift could allow subjects to exercise for longer at the same absolute intensity without the detrimental effects of the anaerobic metabolism. Likewise, it would seem to explain those studies that have demonstrated shorter trial times over a set distance (Peltonen et al. 1995) as it appears subjects could also work at a higher intensity (Byrnes et al. 1984) without elevating levels of fatigue.

The most common effect of HOX reported in the research literature is a decrease in pulmonary ventilation ($\dot{V}_E$) (Asmussen and Nielsen, 1946; Welch et al. 1974; Wilson and Welch, 1975; Ekblom et al. 1975; Byrnes and Mullin, 1981; Plet et al. 1992). This reduction in ventilation is most likely due to a suppression of arterial chemoreceptor responsiveness (Welch et al. 1974; Wilson and Welch, 1975), caused by the higher and more stable partial pressure of arterial oxygen (P_aO_2). A reduction in $\dot{V}_E$ during exercise could have significant implications to firefighting by increasing breathing time on the SCBA bottle and decreasing the hyperventilation of stress (anxiety) and exercise.

Wilson and Welch (1975), proposed that the energy cost of breathing may account for approximately 10% of oxygen uptake at $\dot{V}O_{2max}$. This would suggest that at high workloads, O_2 utilization by the ventilatory muscles may be so great that O_2 supply to the working tissues may be compromised. This would support the findings of Shephard (1966) who postulates that an upper limit for $\dot{V}_E$ during prolonged work is in the range of 120 $l \cdot min^{-1}$ and any increases above this critical value could compromise O_2 delivery to the locomotory muscles due to the exponential rise in the O_2 demand of the respiratory muscles above this point (Robertson et al. 1977). If these findings are accurate then the decrease in $\dot{V}_E$ may be at least partly responsible for the observations of enhanced performance. A decrease in the O_2 demand of the respiratory muscles could make more O_2 available for other physiological processes, including lactate oxidation.

Harms et al. (1997) demonstrated that an increase in the resistance to breathing elevated the O_2 demand of the respiratory muscles and resulted in a 'stealing' of blood flow away from the other working muscles. This finding could be especially relevant during exercise above ventilatory threshold (V_T) while wearing an SCBA. It has been demonstrated that the additional weight of the SCBA (approximately 15.5kg) affects the regulation of breathing and gas exchange (Louhevaara et al. 1985), as well as maximal work capacity (Raven et al. 1977). This is particularly evident at heavier work loads as the straps of the SCBA harness restrict the thorax and increase the work of breathing (W_b). The gas mixture delivered to the firefighter is through a regulator which is designed to pressure assist breathing at low ventilations. However at high ventilations in excess of 100 $l \cdot min^{-1}$, there is a significant inspiratory and expiratory resistance that must be overcome with each ventilatory cycle (Louhevaara et al. 1984). There appears to be a sound theoretical base for the hypothesis that the reduction in ventilation due to HOX, could reduce the ventilatory stress associated with the SCBA. This would also reduce gas consumption and allow firefighters greater time on task and /or a greater safety margin during prolonged operations.

Although the underlying physiological mechanisms are of interest, the two main purposes of this research were to determine whether an increase in F_IO_2 could improve the maximal and submaximal performance of trained SCBA wearers in full firefighting gear.

Only two studies have been attempted in this area, Van den Berg et al. (1977) and Petersen et al. (1999, in press). These studies found that HOX significantly increases recovery and work performance in job specific trials, respectively.

In the study by Van den Berg et al. (1977), firefighters completed a series of treadmill runs while breathing either normoxic (21% O_2) or hyperoxic (40% O_2) gas, delivered in a random fashion. The amount of work completed during the runs was the same in both conditions, but the subjects self-selected the amount of recovery time between the runs. Recovery time was significantly reduced while breathing the hyperoxic gas and the volume of the gas consumed during the hyperoxic trial was significantly less than in the normoxic trial.

Petersen et al. (1999) compared work performance while breathing either 21% (NOX) or 40% oxygen (HOX). Subjects completed a circuit which consisted of five job-related tasks while wearing full turnout gear. The objective was to complete the circuit as fast as possible. Subjects were thoroughly familiarized with the circuit and performance times were reproducible before the experimental trials were undertaken. A randomized double blind, cross-over design was used. Overall time to complete the circuit was significantly decreased and the performance improvements were accompanied by a decreased perception of effort. It is of interest to note that as the circuit was lengthened and the amount of work required was increased (from a mean time of approximately 5 minutes to a mean time of approximately 9 minutes), the performance enhancement effect of HOX was greater.

It appears that HOX could have substantial benefits on the safety and performance of firefighters. However, the limited number of studies looking at the effects of HOX on this unique population have made fairly simple measurements of exercise performance. The aim of this research was to investigate, in a controlled environment, the effects of HOX on the physiological stress associated with maximal and submaximal exercise, while wearing firefighting equipment and breathing through the SCBA.

1.2 Statement of the problem

Previous research has shown that hyperoxia has an ergogenic effect on submaximal endurance performance and during maximal exercise. However, application of this knowledge to applied work situations where a SCBA is normally used to supply air and protect the firefighter from toxic gases is extremely limited (Van den Berg et al. 1977; Petersen et al. 1999). The relevant research findings indicate that HOX could decrease pulmonary, cardiovascular and metabolic stress associated with intense exercise. This is especially important in firefighting since the physiological stresses of exercise are amplified by the restrictions of the protective clothing and SCBA. If HOX had a positive effect, then the benefits could allow firefighters to work for longer duration and/or at a higher intensity than with normoxic air. HOX therefore, has the potential to make firefighting safer and any improvement in physical performance could ultimately save lives and reduce the destruction of property.

1.3 Purpose of the study

The main purpose of this study was to investigate the effects of HOX on selected measurements of physiological and psychophysical stress during maximal and submaximal exercise conditions, while wearing full firefighting gear and breathing through the SCBA. The first condition consisted of graded exercise to exhaustion, which allowed assessment of maximal cardiovascular, pulmonary and metabolic responses. The second condition allowed assessment of submaximal cardiovascular, pulmonary, and metabolic responses, as well as the psychophysical perceptions of exertion and respiratory stress, during 20 minutes of exercise at the ventilatory threshold (V_T).

1.4 Research hypothesis

The following hypotheses were tested:

1. HOX would increase maximal working capacity while wearing firefighting equipment and breathing from a SCBA

2. HOX would decrease ventilatory, cardiovascular and metabolic stress during 20 minutes of strenuous constant-rate exercise at the ventilation threshold (V_T) while wearing firefighting equipment and SCBA.

3. HOX would decrease the psychophysical stress associated with 20 minutes of strenuous constant rate exercise at V_T while wearing firefighting equipment and the SCBA.

1.5 Limitations of the Study

The research might have been limited by

1. Motivation of the subjects to tolerate hard/difficult exercise.
2. All subjects needed to be accustomed to the SCBA, consequently volunteers were invited to participate, rather than being randomly selected.
3. Limited application of the GXT and the experimental trials to 'real' firefighting work.
4. The findings of the research are limited to males.

1.6 Delimitations of the Study

The delimitations of the study were as follows:

1. The study was delimited to twenty-five healthy, male volunteers.
2. The subjects were non smokers of working age (20-55 years) and of moderate to high levels of fitness. Subjects were already accustomed to heavy exercise while wearing the breathing apparatus.
3. The study was delimited to measurements from the specific testing equipment, and the protocols used in the investigation.
4. The subjects completed the six test sessions in a two week period with a minimum of 24 hours rest between tests.
5. Gas mixtures were delivered from identical Scott 4.5 tanks or covered K-size cylinders. This ensured that the F_IO_2 of the gas mixture was unidentifiable to the subjects.

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CHAPTER 2

REVIEW OF LITERATURE

2.1 Introduction

Throughout the literature two main viewpoints have been presented, regarding the limiting factors to maximal aerobic power ($\dot{V}O_{2max}$). One view suggests the limitation lies in the inability of muscle to extract and utilize oxygen. The second view, highlights the inability of the cardiovascular system to meet oxygen demand. It is this second viewpoint that has substantially more support in the literature. However, Wagner (1996) advised that the limitation of $\dot{V}O_{2max}$ may not be uniquely due to cardiac function. Instead he suggested that there is only a partially dependent relationship between $\dot{V}O_{2max}$ and cardiac function and other components throughout the oxygen pathway must also be considered. Wagner (1992;1996) further hypothesized that a reduction in any one of the many factors that influence either pulmonary gas exchange, O_2 delivery, and/or, unloading of oxygen from hemoglobin to the muscle on route to the mitochondria, may determine the maximal power of the aerobic system. This hypothesis appears to combine the central and peripheral limitation theories.

In support of the central limitation hypothesis, HOX has been used to demonstrate that an increase in O_2 delivery can increase maximal aerobic performance (Ekblom et al.1975; Byrnes et al. 1984; Peltonen et al. 1995; Nielsen et al. 1998). Additionally, HOX has also been shown to have an ergogenic effect on performance at submaximal levels (Asmussen et al. 1948; Bannister and Cunningham, 1954; Wilson and Welch, 1975; Plet et al. 1992; Peltonen et al. 1995), which suggests that HOX could have other beneficial effects to the exercising human other than merely improving O_2 delivery during heavy exercise.

2.2 Hyperoxia and Performance

The effect of HOX on $\dot{V}O_{2max}$, has been studied since A.V.Hill and associates first addressed the issue in 1924. The majority of studies have shown that $\dot{V}O_{2max}$ will increase with HOX (Hill et al. 1924; Asmussen and Nielsen, 1958; Ekblom et al. 1975; Linnarsson et al. 1974; Welch et al. 1974; Welch et al. 1977; Weltman et al. 1978; Byrnes et al. 1984; Plet et al. 1992; Peltonen et al. 1995; Nielsen et al. 1998). However, the validity of a number of these studies has been questioned due to difficulties in accurately measuring oxygen consumption.

Research has shown increases in $\dot{V}O_{2max}$ range from a non significant 2% - 4% (Welch and Pedersen, 1981) to a highly significant 22% in a study by Weltman et al. (1978). Due to these discrepancies, it is important to look at the methods that were used. This problem is covered in some detail in the review paper by Welch (1982), who noted that the studies demonstrating the greatest increases in $\dot{V}O_{2max}$ were obtained using Douglas bags for collecting the expired gas. Welch and Pedersen (1981) criticized this method and suggest that the often unavoidable contamination with this method of gas collection can lead to errors possibly as high as 50-75% in $\dot{V}O_2$. These errors have led some researchers to suggest that the measurement of $\dot{V}O_2$ at the mouth is nearly impossible with an F_IO_2 greater than 0.5 (Wagner, 1996). However, Welch and Pedersen (1981) found that the use of rigorously controlled mixing chamber techniques appeared to avoid this contamination and very little variability was found in $\dot{V}O_{2max}$ calculated by three different methods (see chapter 1).

Welch (1982), suggested that even though increases of approximately 10% have been shown (Ekblom et al. 1975; Welch et al. 1977) in the content of arterial oxygen (C_aO_2) with

50-100% O_2, the extraction of oxygen at the muscular level does not increase to the same extent, indicated by the increased P_vO_2 (Welch et al. 1977). Welch, therefore advised that increases in $\dot{V}O_{2max}$ should not exceed 6%. The research done *in situ* by Horstman et al. (1976) on the gracilis muscles of dogs would support this recommendation. A significant increase of 18% was found in C_aO_2 with only a 5% ($p<0.05$) increase in $\dot{V}O_{2max}$ with 100% HOX. However, this finding of Horstman et al. (1976) can probably be at least partially attributed to a significant decrease in $\dot{Q}_{max}$. In disagreement with the findings of Horstman et al. (1976) and Welch et al. (1977), Knight et al. (1993) found that during maximal exercise on a cycle ergometer, 100% O_2 significantly increased leg $\dot{V}O_{2max}$ by 8.1% with an increase in O_2 delivery of 10.9% and no significant change in leg blood flow. The findings from this study therefore, would suggest that some of the assumptions made by Welch, (1982) could be inaccurate, which is certainly possible with the limited amount of published information on blood gases and cardiac output under hyperoxic conditions.

Since 1982 a number of studies that have followed the recommendations of Welch and Pedersen, (1981) have found higher increases in $\dot{V}O_{2max}$ than the theoretical maximum of 6% proposed by Welch (1982). Byrnes et al. (1984); Peltonen et al. (1995) and Nielsen et al. (1998) have demonstrated increases of 11.2%, 13% and 11% in $\dot{V}O_{2max}$, using 70%, 62.2% and 30% O_2, respectively. This would suggest that in human subjects, the use of HOX could increase $\dot{V}O_{2max}$ by approximately 10% assuming that there is no reduction in cardiac output, or blood flow to the working muscles.

A considerable body of evidence also supports the hypothesis that HOX will improve endurance performance, independent of whether the activity is treadmill running (Bannister and Cunningham 1954; Wilson and Welch, 1975), cycling (Asmussen et al. 1948; Asmussen and Nielsen, 1958; Adams and Welch, 1980; Plet et al.1992) or rowing (Peltonen et al. 1995). Some of the original work in this field was by Bannister and Cunningham, (1954) who found that 33-100% O_2 considerably improved time to exhaustion in running performance, often allowing subjects to achieve a steady state at higher intensities than with 21% O_2. These findings are supported by Adams and Welch (1980) and Plet et al. (1992). The latter of these studies, reported a 41% increase in performance time to exhaustion at 80% $\dot{V}O_{2max}$ with 55% HOX. What is interesting in the study of Plet et al. (1992) is that the individual variations, were reported to range from a 21% decrease in time to exhaustion, to a 151% increase. The finding of differing responses to HOX within a relatively homogenous group, raises some interesting questions about the mechanisms responsible for improved performance. Similar findings of a lesser magnitude were also witnessed in the initial study from this institution, where individuals responded differently to HOX during simulated firefighting tasks (Petersen et al. 1999, in press).

Other studies investigating a beneficial effect of HOX on endurance performance, considered whether HOX would allow a set amount of work to be completed in a shorter time, thereby, allowing subjects to work at a higher intensity. These studies also found favourable results. Asmussen et al. (1948) reported that 100% O_2 significantly shortened the time needed to complete a cycling trial, where a low F_IO_2 (12%) increased it. Likewise, Peltonen et al. (1995) reported significantly improved rowing times for a 2500m time trial, with a 62.2% O_2 mixture compared to room air breathing.

One area of study that remains relatively untouched when considering HOX and prolonged submaximal exercise, is whether increasing concentrations of HOX have further performance enhancing effects. In the research by Bannister and Cunningham (1954) it was found that the time to exhaustion increased significantly with increasing F_IO_2 up to 66%. However, there was no further increase in performance with 100% oxygen. Wilson and Welch (1975) disagreed with this finding as they found that 100% O_2 improved performance time significantly over 40% and 60% O_2. Due to these conflicting results and the limited number of studies that have addressed the dose response issue, this is clearly another area that could benefit from more research.

2.3 Potential mechanism for the improved performance with hyperoxia

As previously mentioned the physiological mechanisms responsible for improved performance during maximal and submaximal exercise with HOX are not completely understood. The Fick principle would suggest that for $\dot{V}O_2$ to increase at any intensity of exercise there has to be either an increase in blood flow to the working tissues ($\dot{Q}$), or an increased extraction of oxygen by those muscle (a-vDO_2), or of course, both.

This hypothesis is supported by Ekblom et al. (1975) who found that at maximal exercise the a-vDO_2 was a significant 10% greater with 50% O_2. This occurred with no significant change in cardiac output and a subsequent 11.1% increase in $\dot{V}O_{2max}$. Ekblom and associates (1975) credited this finding to the similar increase in O_2 delivery observed with HOX, which would substantiate the theory that oxygen supply to the working muscle is the limiting factor for performance. This view is supported by Knight et al. (1993), who reported that 100% O_2 increased O_2 delivery by 10.9%, which increased leg $\dot{V}O_{2max}$ by 8.9%.

However, a similar study Welch et al. (1977) found conflicting results, which demonstrate that 60% oxygen breathing did not alter the amount of O_2 consumed by the working muscles during either submaximal or maximal exercise. In this study a 10% increase in the arterial O_2 concentration (C_aO_2) was reported in agreement with the findings of Ekblom et al. (1975) and Knight et al. (1993). However, this increase was accompanied by a significant 11% decrease in leg blood flow and therefore the O_2 supply to the working muscle ($\dot{Q}_x C_aO_2$) remained relatively constant. One criticism of the Welch et al. (1977) study is that the findings were established using repeated measures on two subjects and therefore individual variability may have significantly affected the results.

During submaximal exercise it has been reported that $\dot{V}O_2$ is elevated at submaximal levels with HOX ranging from 40-100% (Hughes et al. 1968; Linnarsson et al. 1974; Wilson et al. 1975). This finding would appear to indicate an increased contribution in ATP supply from the aerobic metabolism. However, the increase in $\dot{V}O_2$ has often been demonstrated without any significant increase in $\dot{V}CO_2$ (Wilson et al. 1975; Welch and Pedersen, 1981; Adams et al. 1986). In fact, two of these studies (Wilson et al. 1975; Adams et al. 1986) report $\dot{V}CO_2$ significantly decreasing. These findings are slightly controversial. It seems more realistic, assuming efficiency isn't affected by HOX, that the energy requirement to work at a similar submaximal level would be the same, independent of F_IO_2 (Hogan et al. 1983). This would agree with the reports of no increase in $\dot{V}O_2$ at submaximal levels found by Welch et al. (1977); Adams and Welch, (1980) and Hogan et al. (1983) and may again highlight the problems of measuring $\dot{V}O_2$ under hyperoxic conditions.

As carbon dioxide is a byproduct of the aerobic metabolism, the lack of an increase in $\dot{V}CO_2$ seems to indicate that the aerobic metabolism hasn't increased with HOX. However, one possible explanation for this is that an increase in O_2 utilization at the muscular level does occur, but that the elevation of P_aO_2 raises the concentration of oxyhemoglobin in venous blood, consequently causing an increased storage of CO_2 via the Haldane effect. Byrnes and Mullin (1981) agreed with this hypothesis and stated that increased storage of CO_2 can be given at least some of the credit for a decrease in $\dot{V}CO_2$ as indicated by the significant increase in end tidal PCO_2 ($P_{ET}CO_2$). However, the study by Wilson et al (1975) found that this was not the case. By estimating P_vCO_2 they found that the maximal storage potential of CO_2 at 80% of $\dot{V}O_{2max}$ was approximately 400ml during 30 minutes of cycling. However, they reported a decrease in $\dot{V}CO_2$ of 4.2 l over the 20 minute trial, with an increase in $\dot{V}O_2$ of 5.6 l with 60% O_2 which would result in a total stored CO_2 closer to 10 litres. This finding appears to greatly exceed the body's maximal storage potential. Therefore, it seems from these calculations that CO_2 storage is not the answer.

The evidence would seem to support no increase in aerobic metabolism during exercise with an increased F_IO_2 and some other explanation is needed. Welch et al. (1974) suggested that some O_2 maybe utilized in other reactions besides those in the electron transport chain. This is supported by Peltonen et al. (1995) who proposed that an increased oxygen utilization for the metabolism of calcium, should not be neglected as a possible factor to explain the increase in $\dot{V}O_2$ and performance.

In accordance with the decrease in $\dot{V}CO_2$ and increase in $\dot{V}O_2$, research with HOX has shown a decrease in the respiratory exchange ratio (RER) (Adams et al. 1986; Hughes et al. 1968; Welch et al. 1974; Welch and Pedersen, 1981). These findings have led some researchers to suggest possible shifts in substrate utilization. Wilson et al. (1975) proposed that there could be a shift from carbohydrate to fat utilization. Welch and Pedersen (1981) agreed with this hypothesis and suggested that the decreased respiratory quotient (RQ) values found at all submaximal levels, indicate a greater use of free fatty acids (FFA), which would be accompanied by an increased oxygen consumption and a decreased $\dot{V}CO_2$, thereby explaining some of the above controversy. However, Plet et al. (1992) found no decrease in RQ values with 55% HOX and suggested that RQ values may not be an accurate indicator of the metabolic state of the muscle in HOX conditions. Therefore, RER values may be of limited benefit in identifying substrate utilization. Furthermore, possible errors in the measurement of $\dot{V}O_2$ in the earlier studies with HOX, also makes the interpretation of RER data from these studies difficult.

Other studies (Miller et al. 1952; Hughes et al. 1968; Ekblom et al. 1975; Adams and Welch, 1980; Knight et al. 1996) have reported decreased concentrations of blood lactate (Bla^-) at maximal exercise, and during submaximal exercise (Ekblom et al. 1975; Wilson et al. 1975; Welch et al. 1977; Knight et al. 1996). These findings appear to support the theory that there is a depression of glycolysis and would also indicate an increased utilization of fatty acid oxidation with HOX. These findings could also explain the decreased $\dot{V}CO_2$, as the decreased Bla^- found during heavy exercise with HOX, would suppress the extra production of CO_2 by bicarbonate buffering (Miyamoto and Niizeki, 1995).

These findings are further supported by Adams and Welch (1980) who reported no significant difference in $[H^+]$ at the end of a significantly longer test to exhaustion with HOX.

The authors suggest that the reduced ventilation and resulting increase in P_aCO_2 with HOX led to an initial increase in $[H^+]$. However, as the duration of the exercise increased, the suppression of glycolysis resulted in a delayed increase in $[H^+]$. This would suggest that during prolonged submaximal work, users of HOX could possibly work longer without the fatiguing effect of a significantly increased $[H^+]$.

Based on the literature reviewed above, there appears to be strong support for a shift in substrate utilization during submaximal exercise, that would allow longer or more intense exercise without the detrimental effects of the anaerobic metabolism. Adams et al. (1986) attempted to substantiate these findings further and hypothesized that if the increase in exercise tolerance with approximately 60% O_2 was due to a substrate shift, then after prolonged submaximal exercise, there should be a decrease in FFA stores and increased blood glycerol levels. This would be without significant changes in either blood glucose or blood alanine concentrations. Nonetheless, the results from their study did not entirely support this hypothesis; alanine levels did remain constant during 30 minutes of exercise at 75% $\dot{V}O_{2max}$. However, no significant changes were found in FFA or glucose values while glycerol significantly decreased. Byrnes and Mullin (1981) also tested a similar hypothesis and found no significant change in muscle glycogen utilization during exercise, which suggested that glycogen sparing may not occur with HOX. Nevertheless, they minimize the importance of their finding by suggesting that a non-significant result may be more indicative of methodological variability, than of muscle glycogen levels. Therefore, although these two studies attempted to substantiate a shift in substrate utilization during exercise by looking at biochemical markers other than Bla^- and $[H^+]$, their findings were inconclusive.

One of the more consistent findings throughout the literature is the decrease in pulmonary ventilation ($\dot{V}_E$) found at submaximal levels with HOX (Asmussen and Nielsen, 1946; Asmussen and Nielsen 1958; Bannister and Cunningham, 1954; Hughes et al. 1968; Ekblom et al. 1975; Byrnes and Mullin, 1981; Peltonen et al. 1995). This decrease in $\dot{V}_E$ has been credited to a depressant effect on chemoreceptor responsiveness as a result of the increased P_aO_2 (Ekblom et al. 1975; Peltonen et al. 1995). A more unusual finding is the reduction in maximal $\dot{V}_E$ by Wilson and Welch (1975) and Ekblom et al. (1975). Ekblom et al. demonstrated that V_E was reduced by 11.1% (17.4 $l \cdot min^{-1}$ BTPS) using a 50% gas mixture. However, no significant difference in $\dot{V}_E$ at maximal levels has been reported by Byrnes et al. (1984); Plet et al. (1992) and Peltonen et al. (1995).

The potential advantages of decreasing $\dot{V}_E$ with respect to firefighting will be covered in more detail later in this literature review. However, from an exercise perspective two main suggestions seem to predominate. Firstly, it is reported that at high work loads the O_2 cost of breathing could account for up to 25% of $\dot{V}O_{2max}$ (Bye et al. 1983) and 14-16% of cardiac output (Harms et al. 1998) which would clearly limit O_2 delivery to other exercising muscles. If this is accurate, then the reduced ventilation with HOX could decreases the O_2 demand of the respiratory muscles, making more O_2 available for the working tissues and other O_2 requiring processes within the body, namely Bla^- oxidation. A further advantage of a reduced $\dot{V}_E$ and a decreased work of breathing (W_b) is addressed by Plet et al. (1992) who demonstrated a 17 $l \cdot min^{-1}$ decrease in $\dot{V}_E$ during heavy exercise and proposed that this could defer respiratory muscle fatigue during endurance events where high ventilations are required for longer periods of time. This assumption is supported by the findings of Aaron et al. (1985) who report that a decrease in the work of breathing with a He-O_2 mixture delayed diaphragmatic fatigue and improved time to exhaustion by 40%.

2.4 Pulmonary function as a limiting factor to exercise

Traditionally, it has been accepted that, pulmonary ventilation is not the limiting factor to endurance performance or $\dot{V}O_{2max}$ and can adequately cope with the demands placed upon it at all intensities of exercise. However, more recently, evidence has been presented that questions this belief. Dempsey et al. (1984) demonstrated that in highly trained athletes ($\dot{V}O_{2max} = 72.2$ ml.kg^{-1}.min^{-1}) P_aO_2 was reduced 21 to 35 mmHg below resting values in 8 out of 16 subjects. A number of factors could be responsible for this exercise induced hypoxemia (EIH). These include venoarterial shunt and ventilation perfusion mismatching (Powers et al. 1993), stress failure of the pulmonary blood gas barrier (Hopkins et al. 1997), diffusion limitation caused by a decreased red blood cell transit time in the pulmonary capillaries and a significant hypoventilation (Dempsey et al. 1984; Power and Williams, 1987; Powers et al. 1993). This decreased alveolar ventilation ($\dot{V}_A$) below the rate needed to maintain an adequate P_aO_2 was discussed by Dempsey et al. (1984) who suggested that a decreased hyperventilatory response within some athletes could be responsible for the decrease in arterial O_2. This is in agreement with Martin et al. (1979) who found a decreased chemoresponsiveness to hypoxia and hypercapnia in endurance athletes and subsequently lower ventilations in athletes compared to non athletes at comparable intensities of exercise.

The occurrence of hypoxaemia to the extent seen in the studies of Dempsey et al. 1984; Powers et al. 1984; Williams et al. 1986; Powers and Williams, 1987; Nielsen et al.

1998, is often only identified in elite endurance athletes (Dempsey et al. 1984; Powers et al. 1993). It would therefore appear that arterial desaturation during high intensity exercise would not be of significance to the majority of firefighters. However, due to the unique physiological responses to maximal exercise while breathing from a SCBA, (Louhevaara et al. 1985) that include a significant hypoventilation, this cannot be total dismissed

There are a number of other well documented factors that could also limit performance from a pulmonary system perspective, that could be accentuated by exercising with the SCBA. At rest the oxygen cost of breathing is approximately 10 $ml{\cdot}min^{-1}$ and during moderate exercise this remains relatively constant at approximately 200 $ml{\cdot}min^{-1}$ (Bartlett et al. 1954). However, during moderate to high intensity exercise the amount of O_2 consumed by the respiratory muscles rises exponentially (Robertson et al. 1977) possibly reaching as high as 8-9 $ml{\cdot}O_2.l^{-1}$ $\dot{V}_E{\cdot}min^{-1}$ (Martin and Stager, 1981). Considering $\dot{V}_E$ can regularly surpass 150 $l{\cdot}min^{-1}$ during maximal exercise the respiratory muscles could demand at least 1200 ml. This increase in the O_2 cost of breathing may be of such an extent that the O_2 supply to the working muscle is compromised (Bye et al. 1983). This hypothesis has been supported by the work of Harms et al. (1997) who demonstrated that a decrease in the work of breathing by a pressure assisted ventilator (PAV) during maximal exercise, reduced the O_2 consumption of the respiratory muscle and resulted in an increase in oxygen and blood flow available for the working muscles. This finding was substantiated further by a considerable 1.3 $l{\cdot}min^{-1}$ decrease in blood flow to the legs during cycling, when the work of breathing was increased by adding mesh screens to the inspiratory line with resistances of 3-5 $cmH_20{\cdot}l^{-1}{\cdot}s$. These findings led Harms et al. (1997) to conclude that the work of breathing during maximal exercise causes a sympathetically mediated peripheral vasoconstriction in the working muscle. This decreases muscle perfusion and hence oxygen supply.

Another possible limitation of the respiratory system that is supported by a growing number of authors is respiratory muscle fatigue (Roussos and Macklem, 1977; Pardy and Bye, 1985; Johnson et al. 1996). This phenomena is defined as the respiratory muscles inability to generate the required pleural pressure (Roussos and Macklem, 1977). As the diaphragm is the largest respiratory muscle and almost solely responsible for inspiratory work; respiratory muscle fatigue is excepted to occur in humans when transdiaphragmatic pressure reaches 40% of maximum (Pardy and Bye, 1985). Tenney and Reese (1968) also demonstrated that the maximum tolerable V_E that can be sustained for a prolonged period of time in normal subjects, is 55% of maximum voluntary ventilation (MVV). Therefore, it appears feasible that respiratory muscle fatigue could occur, as this level of $\dot{V}_E$ is often surpassed during prolonged high intensity exercise. Johnson et al. (1996) suggested that this fatigue appears to be due to an interaction between diaphragmatic pressure production and the aforementioned competition for blood flow, as well as an increase in metabolic byproducts. This is supported by Eldridge (1966) and Jardim et al. (1981) who have demonstrated that sustained breathing against an inspiratory resistance can lead to an increase in [Bla$^-$] produced by the respiratory muscles. However, this appears to be with the exception of the diaphram (Manohar and Hassan, 1990)

Aaron et al. (1985) found that when the work of breathing was decreased by helium breathing (79% He, 21% O_2) in rowers, endurance time increased by 40% during high intensity exercise (90-95% $\dot{V}O_{2max}$). This trend was also shown with lower intensity exercise (80% $\dot{V}O_{2max}$). However, these results were insignificant. He-O_2 has a viscosity of 1.1 times that of air but a density of only 0.34 times that of air (Murphy et al. 1969). Thus, air flow remains laminar at higher intensities with He-O_2, reducing turbulence and airway resistance for a given muscular effort and could therefore reduce the occurrence of respiratory muscle fatigue.

Babcock et al. (1996) demonstrated diaphragmatic fatigue as a result of heavy endurance exercise (treadmill running or cycling at 95% $\dot{V}O_{2max}$ to exhaustion). However, they suggested that the highly fit subjects used in one experimental group were partially protected from excessive fatigue, because of their reduced hyperventilatory response and strengthened diaphragmatic muscles. This finding is a highly relevant one to the present study and may suggest that non-endurance trained individuals maybe more susceptible to respiratory muscle fatigue at high intensities of exercise with the SCBA, which could result in a significantly reduced ventilation and CO_2 retention (Bye et al. 1983).

The concept of respiratory muscle fatigue as a factor in limiting endurance performance has gained further support form a number of studies that have used respiratory muscle training. Boutellier et al. (1992) demonstrated that eight trained subjects improved their breathing endurance from 6.1 to about 40 minutes before the onset of fatigue, following 4 weeks of isolated respiratory muscle training. This improvement in respiratory muscle fitness improved cycle endurance time at the anaerobic threshold (A_T) by 38%. This finding further demonstrates that at relatively high intensities, the ability to perform for long periods of time could be limited by the respiratory system and any decrease in exercise $\dot{V}_E$ at a specific $\dot{V}O_2$, may improve performance.

2.5 The effect of work of breathing on firefighting performance

With respect to firefighting, the increased oxygen cost of breathing and possible respiratory muscle fatigue during high intensity work, is greatly accentuated for two reasons. Firstly, the weight of the self contained breathing apparatus (SCBA) and the harness used to carry it, have been shown (Louhevaara et al. 1985; Raven et al. 1977) to affect gas exchange and to hinder the natural oscillation of the thorax, particularly at high ventilations. Secondly, the compressed air mixture is delivered to the firefighter through a regulator which is subsequently attached to a fitted face piece. This regulator is designed to pressure assist in order to aid breathing at relatively low ventilations (20-40 $l \cdot min^{-1}$). This would theoretically decrease the imposed work of breathing. However, during heavy work where pulmonary ventilation can exceed 100 $l \cdot min^{-1}$, the resistance to breathing is dramatically increased, possibly increasing the work of breathing to levels that could limit performance.

In all the studies that have considered the effects of increased breathing resistance on work tolerance (Cerretelli et al. 1969; Craig et al. 1970; Hermanssen et al. 1972; Demedts and Anthonisen, 1973; Flook and Kelman, 1973; Dressendorfer et al. 1977; Scott Deno et al. 1981), it was found that increasing airway resistance significantly decreases ventilation, $\dot{V}O_{2max}$, and endurance. Although, all studies showed similar findings, it is important to review the possible mechanisms that could decrease an individual's ability to tolerate work under these conditions.

In the most relevant research, Hermanssen et al. (1972) found no significant difference in oxygen uptake at an intensity equivalent to 75% $\dot{V}O_{2max}$ between a conventional valve with an inspiratory and expiratory resistance of 1.7 $cmH_2O{\cdot}l^{-1}{\cdot}s$ and a gas mask (similar to a SCBA), which had an inspiratory resistance of 9 $cmH_2O{\cdot}l^{-1}{\cdot}s$ and an expiratory resistance of 1.7 $cmH_2O{\cdot}l^{-1}{\cdot}s$. However, they did find a 14% decrease in $\dot{V}O_2$ at maximal exercise, which demonstrated that even small increases in breathing resistance can be detrimental to performance.

With an increase in breathing resistance, the human body adjusts to overcome the increased work of breathing by decreasing $\dot{V}_E$ (Hermanssen et al. 1972; Flook and Kelman, 1973; Dressendorfer et al. 1977). In this situation, it has been suggested (Flook and Kelman, 1973) that the body retains CO_2 rather than doing extra respiratory work to maintain adequate $\dot{V}_A$ and CO_2 elimination. This has been supported by the findings of Demedts and Anthonisen, (1973) who showed significant increases in $P_{ET}CO_2$ with increasing exercise load at a high breathing resistance. Killick, (1935) suggested this CO_2 retention could be responsible for the symptoms of distress and the inevitable premature end to exercise that occurs with all increased breathing resistance. However, Craig et al. (1970) only partly agreed and proposed that some other factor is probably more important.

In studies using a number of different resistances (Cerettelli et al. 1969; Dressendorfer et al. 1977; Scott Deno et al. 1981) a decrease in ventilation, $\dot{V}O_{2max}$, and endurance were found, as resistance to breathing increased. Cerrettelli et al. (1969) suggested that the ability of a subject to work was limited before the occurrence of an O_2 debt and therefore, before any energy contribution from the anaerobic metabolism. Craig et al. (1970) agreed that O_2 debt is probably not the limiting factor as there was no difference in O_2 debt found with any of the increased resistances when a comparable amount of work was performed. Cerretelli et al. (1969) go further to suggest that respiratory muscle fatigue cannot be ruled out as a limiting factor to aerobic work. This is again supported by Craig et al. (1970) who theorized that the reduction in endurance with an increased breathing resistance can be attributed to a peculiarity in the ventilatory mechanism rather than a inadequacy in gas exchange.

The resistance to breathing that occurs while breathing at high ventilations through a SCBA is unavailable in the literature. However, it may be concluded that even small increases in breathing resistance can decrease the ability to tolerate work quite extensively. The mechanisms that cause this limitation are not fully understood; but from the previously mentioned literature a decrease in respiratory muscle performance because of the increased work of breathing could be a limiting factor. This is supported by the study of Dressendorfer et al. (1977) who found that 35% O_2 significantly improved work tolerance and $\dot{V}O_{2max}$. The authors attributed this finding to the reduction in ventilation during high intensity work. The evidence therefore suggests that during high intensity work, at or above V_T, while breathing from a SCBA, the resistance to breathing could be a limiting factor and one that could possibly be decreased with HOX.

2.6 Physiological responses to firefighting

Before discussing the potential benefits of HOX on in improving the work tolerance of firefighters, it is important to review the numerous studies that have attempted to assess the physiological stresses involved within this unique occupation.

Firefighting has been demonstrated to be physically demanding (Lemon and Hermiston, 1977a; Lemon and Hermiston, 1977b; Davis et al. 1982; White et al. 1989; Gledhill and Jamnik, 1992). Lemon and Hermiston, (1977a) report that firefighters often work for 1-3 hours at very high intensities (75-95% HR_{max}). These intensities are similar to those found by Davis et al. (1982) who found that during firefighting tasks, heart rates reached an average of 92% $HR_{max.}$

These figures clearly demonstrate a need for a relatively high aerobic capacity to sustain the high intensities of work necessary to fulfill the requirements of the job and also to recover sufficiently when the opportunity arises. Lemon and Hermiston, (1977b) found that the energy cost of four simulated tasks (stair climb, victim rescue, hose drag and ladder raise) ranged from 40.2 to 42.2 $ml{\cdot}kg^{-1}{\cdot}min^{-1}$. Similar findings were also reported by Gledhill and Jamnik (1992) who found that during the most demanding firefighting operations the mean oxygen consumption was 41.5 $ml{\cdot}kg^{-1}{\cdot}min^{-1}$. These findings led to the recommendation that a $\dot{V}O_{2max}$ of 45 $ml{\cdot}kg^{-1}{\cdot}min^{-1}$ was a requirement for firefighters to be able to perform the most intense tasks successfully.

In addition to these high physical demands of firefighting, a number of other stresses have to be considered, namely the SCBA, the thermal properties of the protective clothing and more obviously the environments that have to be worked in.

Firstly, it is important to address the issue of the SCBA. Louhevaara et al. (1985) reported that during treadmill walking at light, moderate and heavy intensities (without the thermal stress of the bunker gear), heart rate was significantly increased at all intensities of exercise. Oxygen consumption and $\dot{V}_E$ were significantly increased at the heavy work load by 0.54 $l \cdot min^{-1}$ and 8.1 $l \cdot min^{-1}$ respectively. However, it should also be mentioned that a significant hypoventilation was seen at the lower intensities. Louhevaara et al. (1985) therefore concluded that the SCBA leads to an insufficient $\dot{V}_E$ and disturbed gas exchange, which are accentuated at the high intensities, mainly due to carrying the extra weight. These findings are consistent with those of Raven et al. (1977) who found that maximal work performance was decreased 18.5% with a similar pressure assisted SCBA. Both of these studies found that the weight of the SCBA (approximately15.5kg) adds to the problems associated with an increased respiratory load that was discussed earlier in this review.

In a study which attempted to look at both the increased physiological stresses of the SCBA combined with the thermal effects of the protective clothing, White et al. (1989) found that wearing full bunker gear and SCBA, significantly increased heart rate and decreased the tolerance time for low intensity work by 85%. This decrease was even greater (96%) during high intensity work. During these work periods skin and core temperature were also significantly higher, with respective 4°C and 0.9°C increases being the greatest differences observed from the control group, who wore light work clothing and no SCBA. These findings document the added stress from the thermal properties of the clothing, even at low to moderate intensities of work (White et al. 1989).

In addition to the aforementioned stresses of carrying and breathing from the SCBA and working within the protective clothing, there is the further added stress of working within very hot environments. Romet and Frim, (1987) commented that the higher heart rates and core temperatures caused by working in protective clothing, are elevated further by the addition of heat exposure, and can eventually become a limiting factor to work performance.

2.6 Hyperoxia and firefighting

Evidence presented in the previous sections supports the ergogenic effect of HOX on performance. However, within elite sport these findings have negligible relevance due to the logistical problems of supplying the large volumes of HOX that would be needed during a race, without carrying the added burden of a SCBA. However, in the very specific occupation of firefighting, or within certain sections of the armed forces, this isn't such a necessary consideration. Within these populations it would be a relatively simple process to change the fraction of O_2 in the tanks that are always worn when entering a toxic or potentially dangerous area.

It is for this reason that it is surprising that the use of HOX as an aid to firefighting performance has not been studied in greater detail. Just two studies can be found within the literature that have addressed this potentially significant area, namely Van den Berg et al. (1977) and Petersen et al. (1999). In the early study, Van den Berg and associates, (1977) found that when running for two minute periods wearing full bunker gear and SCBA, the total resting time needed between bouts of exercise, determined by the firefighters, was reduced by 29% with HOX. This was also achieved with no significant difference in heart rate or ventilation. The latter resulted in a 6% decrease in the amount of air used with HOX, giving good indication that the amount of work that can be achieved on a tank of air, could be increased with an increase in F_IO_2.

In the work by Petersen et al. (1999), the effect of (40%) HOX on the time to complete tasks associated with firefighting, were tested. These tasks have been reviewed in great detail by Gledhill and Jamnik, (1992). Again it was found that HOX improved performance by a 9.6% decrease in the time needed to complete a circuit involving five different firefighting activities. This improvement was associated with no significant difference in heart rate throughout the NOX and HOX trials, which clearly suggests that HOX could reduce at least some of the physiological stress associated with firefighting. This is further supported by the similarity between post exercise blood lactates and a significant decrease in the rating of perceived exhaustion (RPE) towards the end of the test (Petersen et al. 1999).

In conclusion, there seems to be good evidence that HOX can improve the work capacity of firefighters, either by increasing the intensity or duration of work and by decreasing at least some of the physiological stresses that are associated with the job. It appears that the largest potential for HOX could be in the alleviation of respiratory stress which is accentuated by the weight and increased resistance to breathing associated with the SCBA. However, from the presented literature it also appears that HOX may have other potential effects that could greatly improve the safety and performance of those involved within this highly demanding occupation.

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CHAPTER 3

MAXIMAL RESPONSES TO HYPEROXIA WHILE EXERCISING IN FULL FIREFIGHTING GEAR AND SELF CONTAINED BREATHING APPARATUS

3.1 Introduction

Two main viewpoints have regularly been presented in the literature when discussing the limiting factors to maximal aerobic power ($\dot{V}O_{2max}$). One view suggests the limitation lies in the inability of the working muscles to extract and utilize oxygen from the blood (Stainsby et al. 1989; Wagner, 1992). The second, more widely supported view, is the limitation of the central cardiovascular system to transport oxygen to the working muscles (Buick et al. 1980, Dempsey et al. 1984; Rowell, 1986). A more recent hypothesis by Wagner (1992; 1996), proposes that the limitation of $\dot{V}O_{2max}$ may not be uniquely due to cardiac function. Instead, Wagner (1996) hypothesized that there may only be a partially dependent relationship between $\dot{V}O_{2max}$ and cardiac function and a reduction in one of the many factors that either influence pulmonary gas exchange, transport of O_2 to the working muscle, or subsequent unloading of oxygen from hemoglobin to the muscle on the way to the mitochondria, will limit the maximal power of the aerobic system. This concept combines the two previously mentioned hypotheses.

Support for the central limitation hypothesis has often come from hyperoxia (HOX) research, that has used an increased fraction of inspired O_2 (F_IO_2) to enhance O_2 delivery (Ekblom et al. 1975; Horstman et al. 1976). HOX increases the saturation of hemoglobin in arterial blood (S_aO_2) (Knight et al. 1993; Nielsen et al. 1998) and this increases the content of arterial oxygen ($C_aO_2 = 1.39 \times [Hb] \times S_aO_2$), with no significant change in cardiac output

($\dot{Q}$) (Ekblom et al. 1975). This leads to an increase in O_2 delivery ($\dot{Q}$ x C_aO_2). This finding of a similar increase in C_aO_2 and $\dot{V}O_{2max}$ (Ekblom et al. 1975; Nielsen et al. 1998), have been used to demonstrate that when O_2 delivery is elevated, muscular extraction of O_2 increases accordingly (Ekblom et al. 1975; Knight et al. 1993).

While it is generally accepted that HOX has an ergogenic effect on maximal performance, application to sport is generally not feasible. However, this is not the case in occupations that use the self-contained breathing apparatus (SCBA). It is usually accepted that during maximal exercise in normal individuals the pulmonary system will be sufficient to maintain C_aO_2 (Williams et al. 1986). However, it has been shown that the SCBA, hinders the performance of the pulmonary system (Louhevaara et al. 1985), probably due to the weight of the tank, the restrictive nature of the harness and the added resistance to inhaling from the regulator and exhaling through a valve system. These findings could cause an increase in the work of breathing (W_b) and subsequently decrease overall performance by fatiguing the respiratory muscles.

Wilson and Welch (1975) proposed that the energy cost of breathing may account for approximately 10% of oxygen uptake at $\dot{V}O_{2max}$ during normal mouth breathing. This would suggest that at high workloads, O_2 utilization by the ventilatory muscles may be so great that O_2 supply to the working tissues may be compromised. This view is supported by Harms et al. (1997) who demonstrated that an increase in the work of breathing elevated the O_2 demand of the respiratory muscles and resulted in a 'stealing' of blood flow away from the working muscles. It would therefore seem feasible that at high ventilations, the SCBA could amplify any O_2 delivery problems that are present under normal exercise conditions.

The most common effect of HOX is reported to be a decrease in pulmonary ventilation ($\dot{V}_E$) at submaximal work levels (Wilson and Welch 1975; Ekblom et al. 1975; Byrnes and Mullin, 1981; Plet et al. 1992), which is most likely due to a suppression of arterial chemoreceptor responsiveness (Welch et al. 1974; Wilson and Welch 1975), caused by the higher and more stable partial pressure of arterial oxygen (P_aO_2). Harms et al. (1997) found that when the work of breathing was reduced during exercise with a proportional assist ventilator, leg blood flow increased. Therefore, if HOX can 'unload' the respiratory muscles by decreasing $\dot{V}_E$ and the work of breathing, a further enhancement in O_2 delivery might be achieved. This would be of great benefit to SCBA users and could have substantial effects on performance and safety. Therefore, the aim of this study was to investigate the effects of HOX on the maximum performance of subjects wearing full protective firefighting gear and breathing from the SCBA.

3.2 Methods

3.2.1 Subjects

Twenty-five moderately to well-trained non-smoking males provided written informed consent to participate in the study which had previously received institutional ethics review board approval. Twelve subjects were actively employed as firefighters and the remainder were University staff and students. All subjects were completely familiar with breathing through an SCBA during vigorous exercise. Before entry into the study, subjects completed the revised Physical Activity Readiness Questionnaire (rPAR-Q) and in addition, subjects over 40 years old completed a physician-supervised graded exercise test (GXT) with a 12-lead ECG. The physical characteristics (Mean ± SD) of the subjects were as follows: age 32.8 ± 8.9 yr (range: 20 - 52); mass 85.86 ± 9.0 kg (range 67.5 - 100.5); and, height 179.3 ± 5.1cm (range:166 - 189).

3.2.2 Research Design

The experiment was conducted using a single blind, cross-over design where subjects completed three graded exercise tests (GXT) of which one was hyperoxic (GXT_{40}), and two were normoxic ($GXT_{21}P$ and GXT_{21}). Each test was separated by at least 24 hours. The first test ($GXT_{21}P$) served as a practice session to familiarize the subjects with the maximal exercise protocol while dressed in full bunker gear and breathing compressed air with the SCBA. The second test (GXT_{21}), served two purposes: first it allowed the assessment of reliability of exercise responses under normoxic conditions through comparison with $GXT_{21}P$. Secondly, the results were used to evaluate any treatment effect from the hyperoxic gas mixture which was administered during the third test (GXT_{40}). While the tests were conducted in a consistent order, the subjects were led to believe that the order was randomized. Subjects were not informed as to which gas mixture they were breathing during any test until the entire experiment was completed.

Two different methods were used to deliver the inspired gas mixtures. As described below, the first method was used at the beginning of the experiment, but proved to be logistically difficult. The second method permitted several tests to be completed from one cylinder of either compressed air or the hyperoxic gas mixture.

The reliability of the two methods was evaluated with the data from five subjects who each participated in two additional tests (separate from the actual experiment) where the inspired gas was supplied using the methods described below. The order of these tests was randomized and the reliability between the two methods calculated. The results of this procedure revealed that there was no difference in any of the selected physiological ($\dot{V}_E$, $\dot{V}O_2$, $\dot{V}O_2$, HR, RER, SaO_2,) or psychophysical (PRD, or RPE) responses to graded exercise testing between the two methods (Appendix A).

Method 1 : The first 10 subjects were administered normoxic (20.95 ± 0.28%) and hyperoxic (40.64 ± 1.29%) mixtures from identical Scott 4.5 self-contained breathing apparatus (Figure C-1, Appendix C). Gas mixtures were delivered from the SCBA tank carried on the subject's back through a hose coupling, a pressure reducer, and a modified regulator (with the air saver switch, and Vibralert warning devices inactivated).

Method 2 : The remaining 15 subjects had the gas mixtures supplied from two K - size cylinders located adjacent to the treadmill (Figure C-2, Appendix C). The tanks and related fittings were covered so that the subjects could not identify the source of the gas mixture. The gas was supplied to the subject with the same hose, pressure reducer and regulator as described above. The subject carried a half full Scott tank to account for the weight and restriction normally associated with the SCBA and harness. A second pressure reducer was also attached to the harness, since the weight of the one on the breathing line was supported by the handrail of the treadmill.

3.2.3 Graded Exercise Testing

Maximal Oxygen uptake ($\dot{V}O_{2max}$) was determined during progressive walking on a motor driven treadmill at a constant speed and a systematically increasing grade. After being weighed in shorts only, subjects dressed in National Fire Protection Association (NFPA) standard 1500 compliant protective clothing as follows: pants; coat; helmet; flash hood; glove; and, Scott 4.5 SCBA including face piece. Several modifications to the concept of "full protective equipment" were made in order to facilitate data acquisition and/or safety. In the interest of safety during maximal exercise on the treadmill, subjects wore running shoes instead of firefighting boots. In order to allow measurement of S_aO_2 with a finger-tip sensor (pulse oximeter) one glove was not worn. The jacket sleeve above the ungloved hand was pulled up and secured with tape at about mid-forearm to allow access to a catheter which was placed in a distal forearm vein. This also helped to maintain sterility at the blood sampling site. Subjects were weighed again when dressed in full gear to obtain an accurate weight for the protective clothing (21.56 ± 1.46 kg) and also for the calculation of power output.

After a warm-up (5 minutes of walking at 3.5 m.p.h on a level treadmill), subjects stopped walking and performed stretching exercises. The treadmill test speed was set at 3.5 m.p.h. While the treadmill belt was elevated at the rate of 2 % every two minutes. After ventilatory threshold (V_T) had been reached, the treadmill speed remained at the same walking pace, while grade continued to be raised at the rate of 2% per minute until exhaustion. V_T was detected by a systematic increase in the $\dot{V}_E/\dot{V}O_2$ ratio, while the ventilatory equivalent for $\dot{V}_E/\dot{V}CO_2$ remained constant or declined slightly. This "threshold" has been referred to as “Stage II” by Wasserman (1987).

The highest 20 s reading for $\dot{V}O_2$ was accepted as $\dot{V}O_{2max}$ if at least two of the following criteria were met; a plateau in oxygen consumption (increases in $\dot{V}O_2 < 100$ $ml \cdot min^{-1}$) despite an increase in exercise load; respiratory exchange ratio (RER) >1.15; age predicted or previously measured heart rate maximum was achieved; and/or, the subject was too fatigued to continue exercising. Throughout the GXT, heart rate was continuously monitored using a Polar telemetry system (Polar, USA, Inc., Standford, Connecticut)

Respiratory gases were collected using a rigid plastic cone which had been specially designed to fit over the exhalation ports of the SCBA regulator forming an air-tight seal (Figure C-3, Appendix C). The distal end of the cone was linked to the metabolic measurement system using a plastic gas collection hose. Expired gases from the breathing apparatus were collected continuously and gas exchange variables were recorded every 20 s with a SensorMedics 2900z metabolic measurement system (MMC) operating in mixing chamber mode.

Inspiratory and expiratory mask pressure (P_{mask}) was measured in all subjects throughout the GXT. The pressures were measured with a Validyne differential pressure transducer (DP45-28). The transducer was connected to the face piece by a 5mm inside diameter hose, attached to a specifically designed metal disc with a small metal nipple on the exterior surface. The disc replaced the cover of the voice port of the mask, creating an air tight seal. The pressure transducer was connected to a Validyne (CD19) amplifier which was attached to a chart recorder (General Scanning, RS6 5-P). Inspiratory and expiratory mask pressures were recorded for the last 15 seconds of every work load, and at maximal exercise.

Throughout GXT_{21} and GXT_{40} a blood sample of approximately 1 ml was taken from an indwelling 22 gage teflon intravenous catheter which was inserted into a convenient vein in the distal forearm. A 'Hi-Flo' three way stopcock (Medex, Hillard, Ohio) was attached to the catheter and the whole device was taped to the arm or the back of the hand. Blood samples were taken at the intensity which elicited V_T in $GXT_{21}P$ (VT_N) and the end of the workload immediately following VT_N. A third sample was taken immediately after the test was terminated. Subjects then performed an active recovery (a slow walk standardized for all subjects at 2.5 m.p.h on a level treadmill) for 3 minutes at which time the treadmill was stopped and another sample was taken 2 min later, exactly 5 minutes following the end of the test.

Immediately after sampling, 200μl of blood were transferred to a preservative tube (Yellow Springs Instruments, YSI 2315). The tube was vortexed to mix the whole blood and preservative powder. The preservative tubes were stored at -20°C until lactate analysis was performed. The analysis was completed within three weeks of the sample being taken. All samples were measured in duplicate with a YSI Model 27 analyzer. Calibration of the analyzer was verified with known standards between each set of duplicates.

At the end of each workload during the GXT, subjects were asked to indicate a numerical rating from the 15-point Borg Scale (Borg, 1982) which most closely matched how they perceived the 'overall' stress of the exercise. In conjunction with the rating of perceived exertion (RPE), perceptions of respiratory distress (PRD) were also measured using a 7 point psychophysical scale developed by Morgan and Raven (1985). Subjects were asked to indicate the rating which most closely matched their perception of breathing difficulty and toward maximal exercise this also indicated whether the subjects perceived the air supply to be sufficient.

3.2.4 Statistical Analysis

All dependent variables from the two experimental conditions, namely hyperoxia (40%) and normoxia (20.93%) and between tests ($GXT_{21}P$, GXT_{21} and GXT_{40}) were analyzed using a repeated measures analysis of variance (ANOVA). A post hoc multiple comparisons test, the Scheffe-procedure, was used if appropriate. The alpha level was set at 0.05 for both the analysis of variance and the post hoc test.

3.3 Results

The reproducibility of all measurements taken throughout the graded exercise tests are presented in Table 3-1. No significant difference was found for $\dot{V}O_2$, $\dot{V}CO_2$, RER, $\dot{V}_E$, HR, or S_aO_2 between $GXT_{21}P$ and GXT_{21}. However, it was felt that the variables from the GXT_{21} test, which was administered following the practice test $GXT_{21}P$, were "truer" maximal values and would have accounted adequately for learning effects. This conclusion was supported by a significant increase (16W) in the maximal power output reached by the 25 subjects in GXT_{21} with no further increase in $\dot{V}O_{2max}$. Therefore, the data from GXT_{21} were used for comparison with the hyperoxia condition.

Table 3-2 shows that 40% O_2 increased ($p<0.05$) $\dot{V}O_{2max}$ from a mean value of 4.544 $l\cdot min^{-1}$ to 5.002 $l\cdot min^{-1}$ (Figure 3.1). However, it should be mentioned that no significant difference in $\dot{V}O_2$ was observed in GXT_{40} until a power output of greater than 90% of normoxic power output max (NPO_{max}) had been exceeded (Figure 3.2). The 10.1% increase in $\dot{V}O_{2max}$ was accompanied by a significant increase of similar magnitude in $\dot{V}CO_2$ (9.2%). Maximal $\dot{V}_E$ and maximal heart rate were the same, irrespective of the gas mixture (Figures 3.3 and 3.4).

Coincident with the increase in $\dot{V}O_{2max}$, a significant increase in maximal power output was observed in the HOX condition. On average, power output was increased by 33W (321W in GXT_{21} compared to 354W in GXT_{40}) before volitional exhaustion resulted in the termination of the test (see Figure 3.5). It should also be noted that HOX did not affect the ratio of $\dot{V}O_{2max}$ and power output. The VO_2 required to generate 1 W of power was 14.18 ± 0.8 ml and 14. 20 ± 1.1 ml in the NOX and HOX conditions, respectively.

Figure 3.6 shows that a significantly lower ($p<0.05$) S_aO_2 value was observed at $\dot{V}O_{2max}$ in GXT_{21}. The mean pre-test value for S_aO_2 was 96.3% which dropped to 91.3% at normoxic $\dot{V}O_{2max}$ ($N\dot{V}O_{2max}$). However, in the hyperoxic condition, two differences were observed. First, S_aO_2 was significantly higher at rest, and second, the decline in S_aO_2 during the test was considerably less than observed during GXT_{21} pre-test level of 98.8% to 97.5% at hyperoxic $\dot{V}O_{2max}$ ($H\dot{V}O_{2max}$). There was a significant decrease in S_aO_2 between rest and $H\dot{V}O_{2max}$.

Mask pressure was successfully measured in 18 of the 25 subjects at $N\dot{V}O_{2max}$ and $H\dot{V}O_{2max}$. The values obtained for P_{mask} on inspiration and expiration at $\dot{V}O_{2max}$ in NOX and HOX were not significantly different (Table 3-3). Inspiratory mask pressure was -10.46 and

-10.32 cm H_2O, and expiratory mask pressure was 13.24 and 12.79 cm H_2O, for NOX and HOX, respectively. It was observed that the negative P_{mask} generated on inspiration was of a significantly lower magnitude than the positive P_{mask} necessary for expiration.

At the power output that elicited $N\dot{V}O_{2max}$ a lower ($p<0.05$) inspiratory P_{mask} and expiratory P_{mask} were found with HOX (Table 3-3). This reduction in P_{mask} was accompanied by a significant reduction in $\dot{V}_E$ (191.6 compared to 172.7 $l \cdot min^{-1}$ in NOX and HOX, respectively). The combination of these two findings (a reduction in P_{mask} and $\dot{V}_E$) at $N\dot{V}O_{2max}$ with HOX, suggests that during high intensity exercise the W_b was also reduced.

Table 3-4 illustrates the blood lactate concentrations ($[Bla^-]$) at selected time intervals for GXT_{21} and GXT_{40}. Samples were successfully obtained from 22 subjects and results are displayed graphically in Figure 3.7. There was a reduction ($p<0.05$) in blood lactate concentrations from samples taken at the two submaximal levels. In contrast, the lactate samples drawn at the termination of the tests and after 5 minutes of standardized recovery were not significantly different between the NOX and HOX conditions. However, it should be recalled that a higher power output was achieved with HOX.

Table 3-1: Reproducibility between tests for the normoxic graded exercise tests ($GXT_{21}P$ and GXT_{21}).

	$GXT_{21}P$	GXT_{21}
$\dot{V}O_2$	4.519 ± 0.80	4.544 ± 0.81
$\dot{V}CO_2$	5.216 ± 0.10	5.343 ± 0.10
RER	1.17 ± 0.10	1.19 ± 0.11
$\dot{V}_E$	184.7 ± 4.46	189.0 ± 3.39
HR	189.0 ± 2.33	190.0 ± 2.12
SaO_2	91.3 ± 0.57	91.3 ± 0.40
PO	305 ± 7.25	321 ± 6.46*

Values are means ± SE for 25 subjects. Oxygen consumption ($\dot{V}O_2$) measured $l \cdot min^{-1}$ (STPD), carbon dioxide produced ($\dot{V}CO_2$) measured in $l \cdot min^{-1}$ (STPD), ventilation ($\dot{V}_E$) measured in $l \cdot min^{-1}$(BTPS), heart rate measured in b·p·m, arterial saturation (SaO_2) measured as a percentage and power output (PO) measured in W. * = $p<0.05$.

Table 3-2: Comparison of maximal values measured during graded exercise tests under normoxic and hyperoxic conditions (GXT_{21} and GXT_{40}).

	GXT_{21}	GXT_{40}
$\dot{V}O_2$	4.544 ± 0.81	5.002 ± 0.10*
$\dot{V}CO_2$	5.343 ± 0.10	5.834 ± 0.11*
RER	1.19 ± 0.11	1.18 ± 0.01
$\dot{V}_E$	189.0 ± 3.39	186.9 ± 3.41
HR	190.0 ± 2.12	191.4 ± 2.00
SaO_2	91.3 ± 0.40	97.5 ± 0.157*
PO	321 ± 6.46	354 ± 8.54*

Values are means ± SE for 25 subjects. Oxygen consumption ($\dot{V}O_2$) measured $l \cdot min^{-1}$ (STPD), carbon dioxide produced ($\dot{V}CO_2$) measured in $l \cdot min^{-1}$ (STPD), ventilation ($\dot{V}_E$) measured in $l \cdot min^{-1}$(BTPS), heart rate measured in b·p·m, arterial saturation (SaO_2) measured as a percentage and power output measured in W. * = $p<0.05$.

Table 3 : Comparison of mask pressure and ventilation values measured during graded exercise tests under normoxic and hyperoxic conditions (GXT_{21} amd GXT_{40}).

	GXT_{21}	GXT_{40}
I_{max}	-10.46 ± 0.45	-10.32 ± 0.41
E_{max}	13.24 ± 0.63	12.79 ± 0.66
$I@N_{max}$	-10.46 ± 0.45	-8.07 ± 0.37 *
$E@N_{max}$	13.24 ± 0.63	10.67 ± 0.77 *
$\dot{V}_{E\ max}$	191.6 ± 3.64	189.1 ± 3.69
$\dot{V}_E@N_{max}$	191.6 ± 3.64	172.7 ± 3.82 *

Values are means ± SE for 18 subjects. Mask Pressure during inspiration at maximal exercise (I_{max}) measured in cmH_2O, mask pressure during expiration at maximal exercise (E_{max}) measured in cm H_2O, mask pressure during inspiration at the intensity that elicited normoxic $\dot{V}O_{2max}$ ($I@N_{max}$) measured in cmH_2O, mask pressure during expiration at the intensity that elicited normoxic $\dot{V}O_{2max}$ ($E@N_{max}$) measured in cmH_2O, maximal ventilation ($\dot{V}_{E\ max}$) measured in $l \cdot min^{-1}$ and ventilation at the intensity that elicited normoxic VO_{2max} ($\dot{V}_E@N_{max}$) measured in $l \cdot min^{-1}$ (BTPS). * = $p<0.05$.

Table 3-4 : Comparison of blood lactate responses during graded exercise tests under normoxic and hyperoxic conditions (GXT_{21} and GXT_{40}).

	Samples			
	1	2	3	4
NOX_{21}	2.42 ± 0.10	2.94 ± 0.13	7.78 ± 0.40	11.27 ± 0.36
HOX_{40}	1.86 ± 0.08*	2.25 ± 0.08*	8.14 ± 0.41	11.25 ± 0.34

Values are means ± SE for 22 subjects. Blood lactate measured in $m \cdot mol^{-1} \cdot l^{-1}$. * = $p<0.05$.

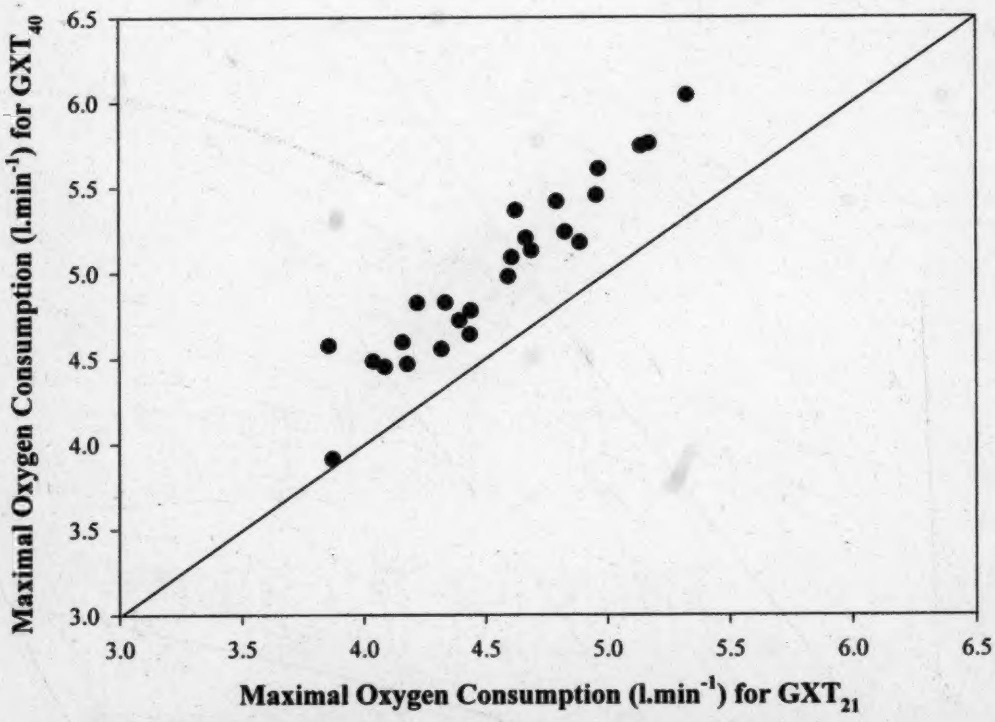

Figure 3.1 : Maximal oxygen consumption responses (n=25) measured during GXT$_{21}$ and GXT$_{40}$.

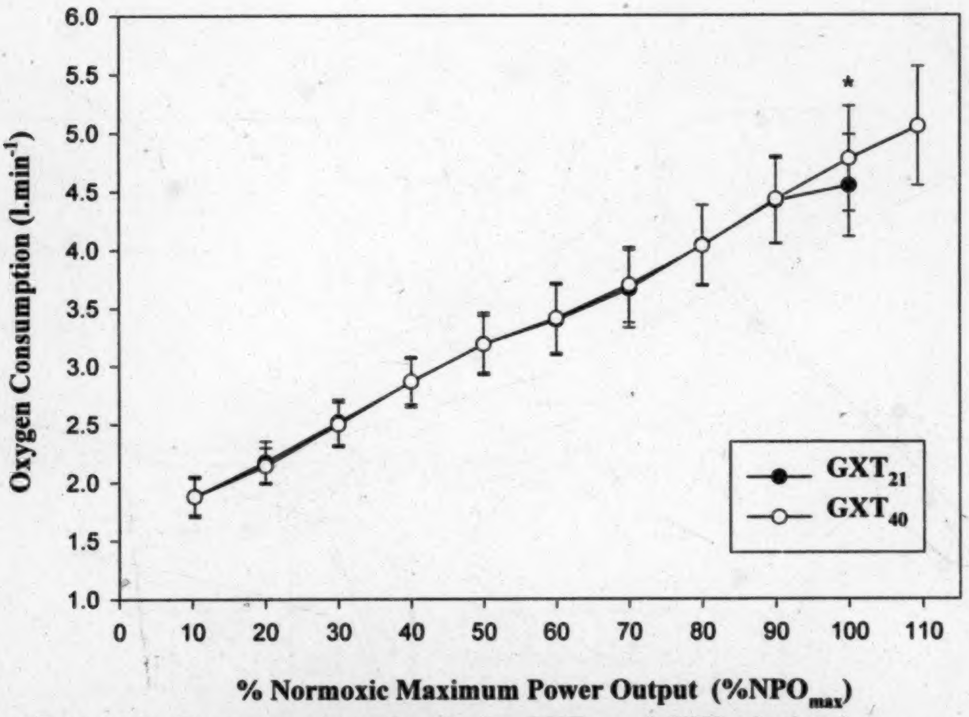

Figure 3.2 : Oxygen consumption during GXT_{21} and GXT_{40} depicted as a percentage of normoxic maximum power output. n=25, * = p<0.001. Hyperoxic VO_{2max} is a representation of the 17 subjects who achieved a higher power output compared to NPO_{max}.

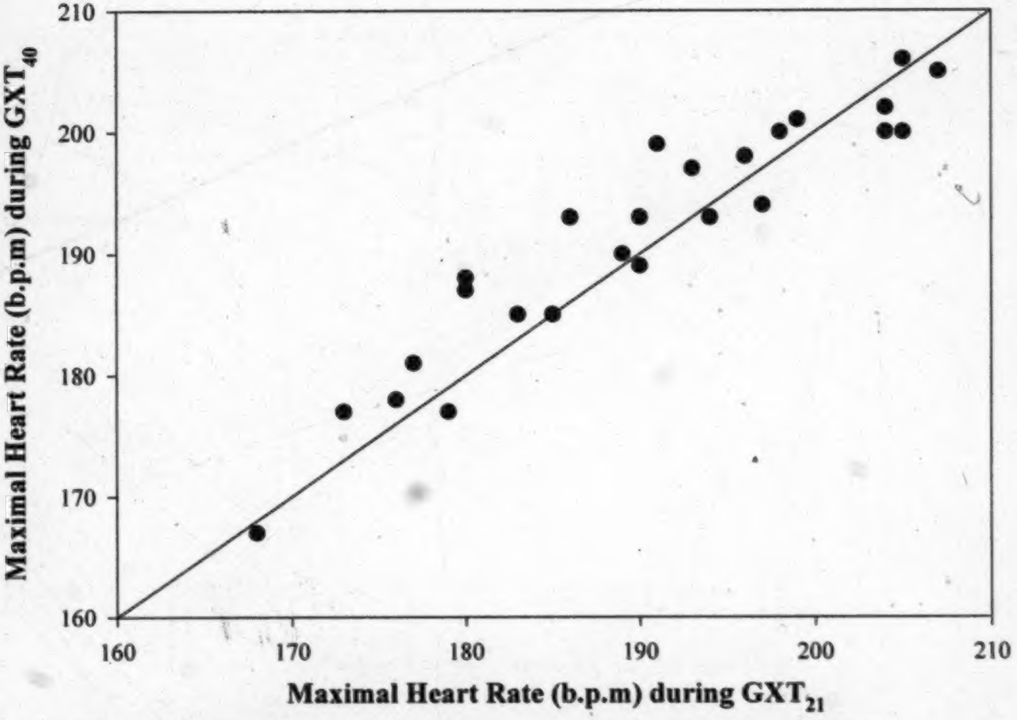

Figure 3.3: Maximal heart rate responses during GXT_{21} and GXT_{40} n=25.

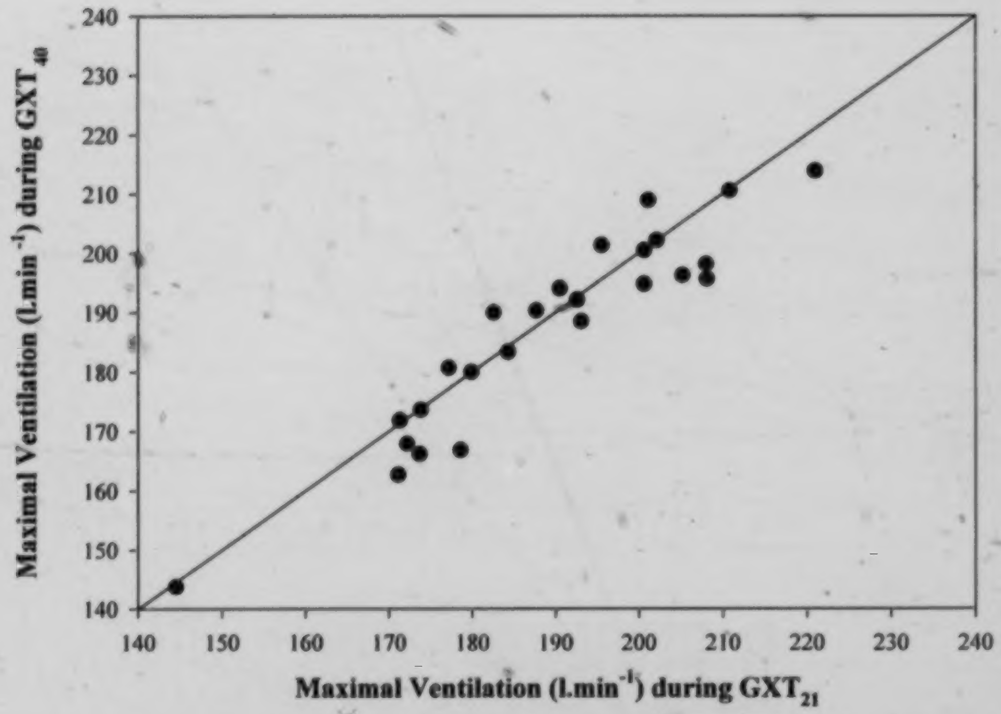

Figure 3.4 : Scatterplot of maximal ventilation during GXT_{21} and GXT_{40}. n=25.

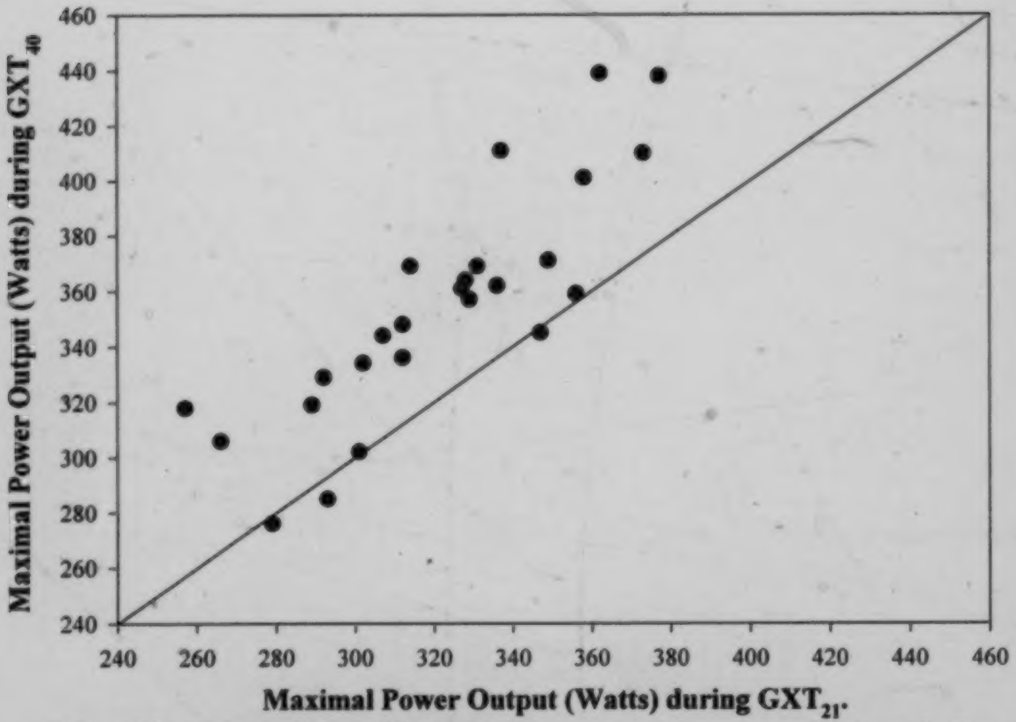

Figure 3.5 : Scatterplot (n=25) showing the maximal power output reached during GXT_{21} and GXT_{40}.

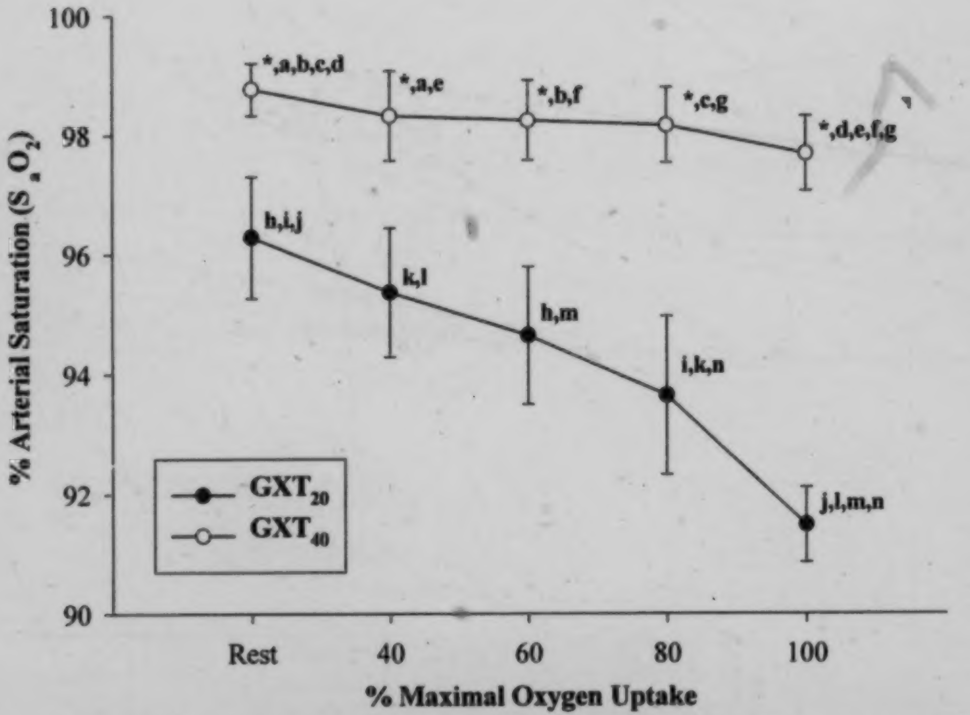

Figure 3.6 : Arterial saturation depicted as a percentage of VO_{2max} during the two GXT_{21} and GXT_{40} (n=25). * =indicates significant difference between GXT_{21} and GXT_{40} ($p<0.05$). Paired lower case letters indicate significant differences ($p<0.05$) within a test.

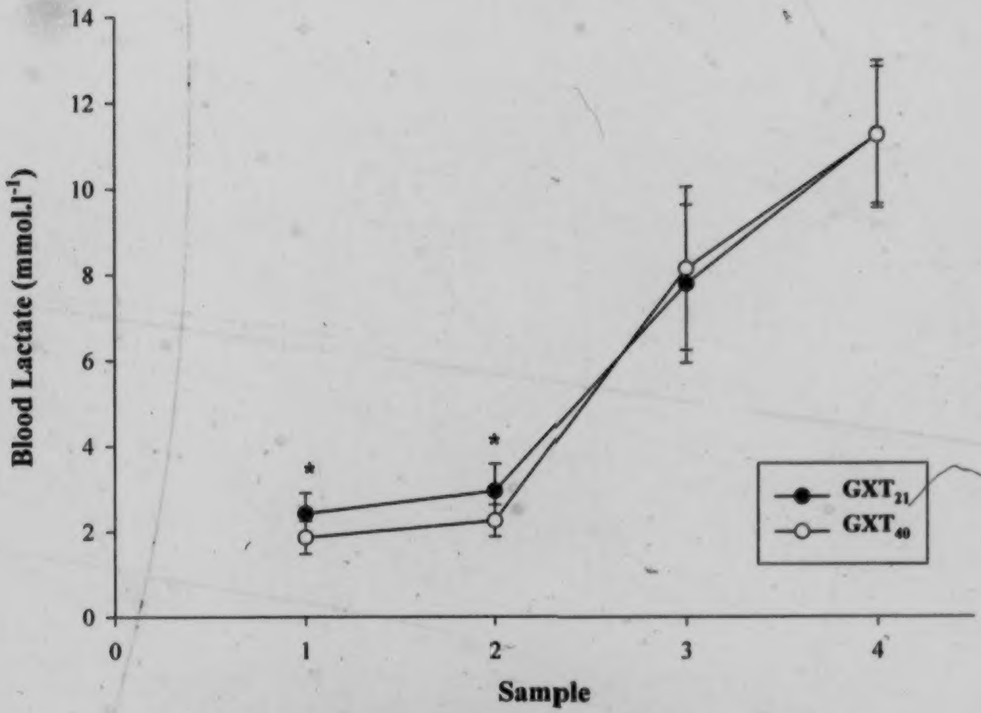

Figure 3.7 : Blood lactate responses during GXT_{21} and GXT_{40}. n=22. * = $p<0.05$.

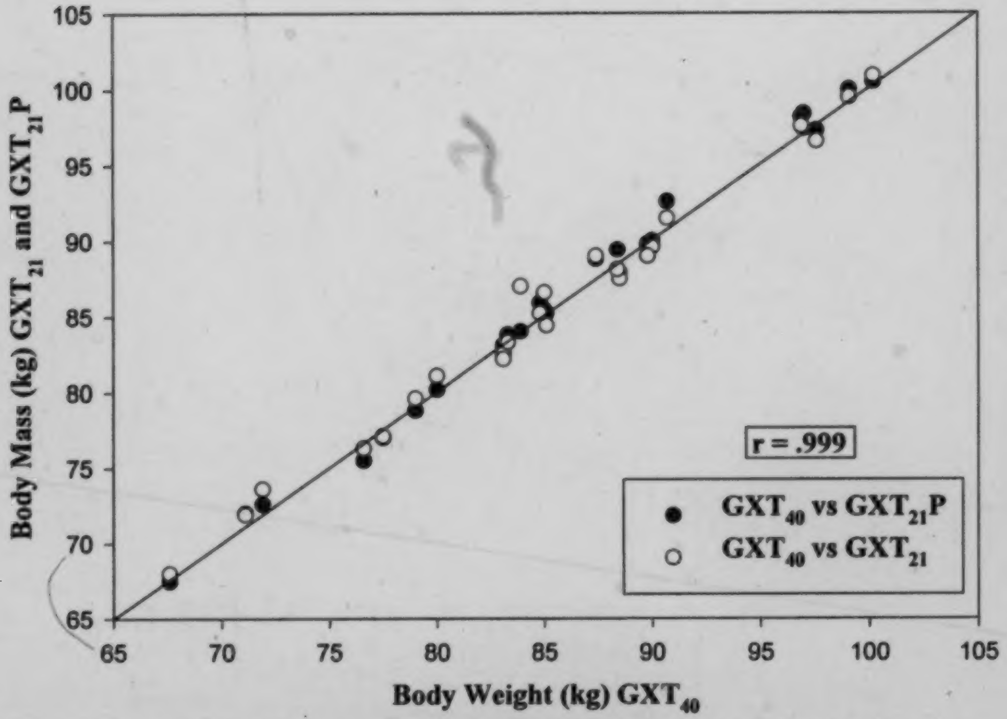

Figure 3.8: Scatterplot demonstrating the reliability of subject weight for the three graded exercise tests ($GXT_{21}P$, GXT_{21} and GXT_{40})

3.4 Discussion

The main finding of this investigation was a 10.1% increase in $\dot{V}O_{2max}$ with HOX , which is in agreement with the findings of previous research (Hughes et al. 1968; Ekblom et al. 1975; Byrnes et al. 1984; Peltonen et al. 1995; Nielsen et al. 1998) with O_2 mixtures ranging from 30-70%. The increase in $\dot{V}O_{2max}$ was accompanied by increases in maximal power output and $\dot{V}CO_2$ of a similar magnitude (9.3% and 9.2%, respectively) However, at the increased $\dot{V}O_{2max}$ and power output, no significant differences in maximal HR, $\dot{V}_E$, [Bla$^-$], or RER were observed between GXT_{21} and GXT_{40} .

The oxygen cost of exercise ($\dot{V}O_2$) at each progressive workload should be the same for both GXT_{21} and GXT_{40} as no difference in body mass was observed between the tests (Fig 3.8). The data in Fig 3.2 show that $\dot{V}O_2$ was not significantly different at any submaximal workload up to 90% of the NPO_{max}. This finding is supported by other research (Hogan et al. 1983; Adams and Welch, 1980) and demonstrates that when a mixing chamber is used for expired gas collection (as recommended by Welch and Pedersen, 1981) $\dot{V}O_2$ should not be elevated at submaximal levels. It also indicates that our measurement system was well calibrated and not significantly affected by the higher F_IO_2 (Eccles et al. 1986).

Arterial saturation was significantly decreased at $\dot{V}O_{2max}$ compared to resting values in both GXT_{21} and GXT_{40}. However, the decrease in S_aO_2 with HOX was considerably less than with NOX . This agrees with the findings of Nielsen et al. (1998) and demonstrates that 40% O_2 can help maintain hemoglobin saturation. Hemoglobin saturation decreased significantly throughout GXT_{21} to a minimum value of 91.5 ± 2.2% at $N\dot{V}O_{2max}$. The decrease in S_aO_2 in healthy individuals during maximal exercise is well documented in the literature (Dempsey et al. 1984; Powers et al.1984; Williams et al.1986; Powers and Williams,1987; Nielsen et al. 1998). Dempsey et al. (1984), identified exercise induced hypoxaemia (EIH,) in 12 of 16 highly trained subjects ($\dot{V}O_{2max} = 72 \pm 2$ ml.kg^{-1}min^{-1}). The criteria used for the identification of EIH was a 10-35 mmHg reduction in P_aO_2, which unfortunately we cannot compare our findings to, as blood gas parameters were not measured. However, the mean S_aO_2 found on completion of $GXT_{21,}$ is comparable to the 91.9% and 92.9% reported by Dempsey et al. (1984) and Nielsen et al. (1998) respectively. It would therefore seem possible that EIH and a subsequent decrease in O_2 delivery were limiting factors to a number of our subjects, especially as seven of the 25 subjects had S_aO_2 values of 90% or lower at $\dot{V}O_{2max}$.

There are a number of mechanisms that could be responsible for EIH, including ventilation-perfusion inequality, hypoventilation, and venoarterial shunt (Powers and Williams, 1987), and possibly, stress failure of the pulmonary blood gas barrier (Hopkins et al., 1997). However, the most likely mechanism in well trained individuals, seems to be pulmonary diffusion limitation, caused by a reduced red cell transit time within the pulmonary capillary. Dempsey et al. (1984) suggested that if pulmonary capillary transit time is lowered to below 0.4s EIH is possible. The authors estimated that for this phenomena to occur, pulmonary capillary blood volume must be maximized, which would require a cardiac output in excess of 26 l·min^{-1} and a $\dot{V}O_2$ in the range of 4.0-5.0 l·min^{1}. Therefore, it seems possible that a limitation in pulmonary diffusion could be responsible for the desaturation observed within our subjects, as the mean $\dot{V}O_{2max}$ was 4.54 ± 0.81 l·min^{1} in

GXT_{21}.

Nevertheless, as our subjects were not as highly trained as Dempsey et al's., subjects ($\dot{V}O_{2max}$ 53.4 vs 72 ml·kg·min^{-1}), it seems unlikely that they elicit cardiac outputs sufficient to decrease pulmonary capillary transit time to an extent that would decrease S_aO_2. However, there are two other possible explanations for the EIH. Firstly, the ventilatory response to heavy work during NOX in our subjects may be affected by the increased mechanical load placed on the chest wall by breathing from the SCBA. The SCBA presents a unique condition for exercise, especially in relation to pulmonary mechanics. It has been demonstrated (Louhevaara et al. 1985; Raven et al. 1975) that the natural oscillation of the thorax and the resulting gas exchange are hindered by the weight and restriction of the SCBA shoulder straps and tank. The SCBA regulator is designed to pressure assist on inspiration at low ventilations. However, during maximal exercise in our subjects, where pulmonary ventilation reached levels above 180 l·min^{-1}, it appears that the resistance to breathing is dramatically increased, as demonstrated by the increase in P_{mask} on both inspiration and expiration. It seems feasible that the restriction placed on the chest wall combined with an added resistance to breathing, could substantially increase the elastic and resistive components of breathing, respectively. This could lead to a diminished hyperventilatory response and a decline in oxygenation, especially at workloads above V_T. However, it is difficult to make any substantial conclusions, as the ventilatory responses of our subjects during a GXT without the SCBA, were not assessed in this study.

A second factor that could contribute to the reduction in S_aO_2 is an accentuated Bohr effect. Exercising in protective clothing has been shown to increase core temperature

(Duncan et al. 1979; Smith et al.1997) as thermoregulation is restricted. In addition, an increase in metabolic acidosis, shown by the high post-exercise [Bla^-], would decrease blood pH. These two factors in combination could displace the O_2 disassociation curve further to the right than in normal exercise, resulting in a greater unloading of O_2 at the muscle and a decreased P_VO_2. If this decreased P_VO_2 is then combined with a ventilatory limitation due to mechanical constraint of the SCBA, insufficient oxygenation of venous blood could occur. This would be accentuated further by a decreased O_2-loading at the lung with the increased acidosis. The partial pressure of alveolar O_2 (P_AO_2) has been shown to significantly increase with HOX (indicated by the elevated $P_{ET}O_2$ reported by Nielsen et al.1998), which reduces the gradient across the alveolar-arterial membrane and appears to protect subjects against desaturation, even though [Bla^-], $\dot{V}_E$ and P_{mask} were similar on completion of the two GXTs.

The increase in $\dot{V}O_{2max}$ was accompanied by an increase in maximal power output during GXT_{40}, which disagrees with previous studies which found no increase in PO_{max} (Nielsen et al. 1998) and no significant increase in either $\dot{V}O_{2max}$ or PO_{max} (Hogan et al. 1983). Hogan et al. (1983) reported that $\dot{V}O_{2max}$ only varied when a higher maximal work rate was attained. However, in this study seven of our subjects achieved a higher $\dot{V}O_{2max}$ in GXT_{40} at the same maximal power output. There are two explanations for this. Firstly, 12 subjects elicited a plateau in oxygen consumption (identified by <100ml increase in $\dot{V}O_2$ for an additional increase in power output) during GXT_{21}, which was not apparent in any of the subjects of Hogan et al. Secondly, in GXT_{21} subjects were encouraged to attempt further workloads, even if only 20s (one reading on the 2900z MMC) of the higher work load could be attained before exhaustion. With HOX, subjects would often complete this unfinished workload and an increase in $\dot{V}O_{2max}$ would be observed, even though subjects failed to increase power output.

Nielsen et al. (1998) observed that the maintenance of S_aO_2, stabilized P_aO_2, subsequently increasing C_aO_2 by 8.5 % and $\dot{V}O_{2max}$ by 11 ± 3%. However, as mentioned above, Nielsen et al. did not find an increase in maximal power output. These authors postulated that O_2 delivery ($\dot{Q} \times C_aO_2$) must have remained constant, probably due to the vasoconstriction caused by HOX, with a decrease in blood flow to the working muscles (Welch et al. 1977). However, Knight et al. (1993; 1996) have demonstrated that leg blood flow ($\dot{Q}_{legs}$) did not decrease with 100% O_2 during maximal exercise on a cycle ergometer. This finding was supported further by Richardson et al., (1998), who suggested that an increased PO_2 may not have a vasoconstrictor effect on muscle arterioles during heavy exercise. These authors hypothesized that the increased metabolic demand at the mitochodrial level may nullify the effect of the high PO_2 on vascular conductance and subsequently diminish the local autoregulatory signal to vasoconstrict.

Therefore, it would seem plausible that if there is a compensation for the vasoconstricting effect of HOX, O_2 delivery would increase. This postulate is supported by Ekblom et al. (1975) who reported that breathing 50 % O_2 increased P_aO_2 to 260mmHg and increased C_aO_2 by 7.7% with no change in cardiac output. Ekblom et al. (1975) hypothesized that this increased C_aO_2 led to a significant increase in O_2 delivery and a increase in the a-$\bar{v}DO_2$ of 11.2%, which would explain the majority of their 12.6% increase in $\dot{V}O_{2max}$. These findings further substantiate the hypothesis of an O_2 delivery limitation to maximal aerobic exercise which is supported by our similar increases in $\dot{V}O_{2max}$ and PO_{max} in GXT_{40} and the 'leveling off' of $\dot{V}O_2$ with a decrease in S_aO_2 in GXT_{21}.

The recent study of Richardson et al. (1998) also provides substantial support for the O_2 delivery limitation hypothesis. These authors concluded that when convective arterial O_2 delivery was kept constant, and the O_2 disassociation curve was artificially shifted to the right using an allosteric modifier of Hb (RSR-13, Allos therapeutics), the subsequent shift in P50 (PO_2 at which 50% Hb is saturated) increased VO_{2max} by 20% in electrically stimulated dog muscle. These findings clearly demonstrate that O_2 delivery is the limiting factor to exercise and with a greater PO_2 gradient from capillary to muscle, a-vDO_2 will increase, as demonstrated with HOX (Horstman et al. 1976; Ekblom et al. 1975). Although O_2 delivery can be the limiting factor, an exception to this hypothesis may exist. In the present study increases in $\dot{V}O_{2max}$ ranged from 1.1% - 15.7%, and clearly some subjects have a greater response to an elevated F_IO_2 than others. Richardson et al. (1998) reported 'non responders' to an artificial shift in the O_2 dissociation curve and suggested that the dogs that didn't respond may already have reached their mitochondrial metabolic potential. If that were the case, an increase in O_2 delivery may only have a minimal effect on subjects who are already close to maximal extraction. The concept of mitochondrial metabolic reserve is an interesting one which warrants further research.

No significant difference in maximum $\dot{V}_E$ or P_{mask} was found at $\dot{V}O_{2max}$ in GXT_{21} and GXT_{40}. However, at $N\dot{V}O_{2max}$, significant reductions in $\dot{V}_E$ (9.9%) and P_{mask} (20.9%) were found across the ventilatory cycle with HOX. The combination of these findings suggests that there was a significant reduction in the work of breathing (W_b) during GXT_{40} at higher pulmonary ventilations. Harms et al. (1998) have demonstrated that during maximal exercise, the respiratory muscles demand 14 - 16% of maximum cardiac output. In order to satisfy this demand, Harms et al. (1997) reported that blood flow to the working muscles is 'stolen' from the legs, due to a sympathetically mediated peripheral vasoconstriction that reduces blood flow. If W_b is increased with the SCBA, then respiratory muscle demand will be elevated even further requiring an greater proportion of the cardiac output. Logically, if HOX can 'unload' the respiratory muscles, the reduction in W_b could conceivably increase $\dot{Q}_{legs}$, O_2 availability, and ultimately $\dot{V}O_{2max}$.

These findings help explain the decrease in [Bla^-] found at submaximal levels in GXT_{40}, at and above V_{TN}. Jardim et al. (1981) and Eldridge (1966) have shown that sustained breathing against an inspiratory resistance can increase the amount of lactate produced by the respiratory muscles, with the exception of the diaphram (Manohar and Hassan, 1990). A decrease in the W_b would likely decrease anaerobic work in the respiratory muscles, which could result in a lower [Bla^-]. Additionally, if pulmonary blood flow is decreased then more blood flow would be available for other tissues in the body other than the working muscles. Adams et al., (1986) suggested that HOX may enhance splanchnic blood flow which would enhance the liver's uptake of Bla^- and help explain the lower submaximal [Bla^-] found in GXT_{40}.

In summary, HOX significantly increased $\dot{V}O_{2max}$ and maximum power output and appears to protect S_aO_2 at high work intensities. The decreased $\dot{V}_E$ and P_{mask} at submaximal intensities with HOX suggest that W_b is also significantly decreased, which could alter blood flow to the working muscles. This finding, in conjunction with an elevated S_aO_2, would lead to an increase in O_2 delivery which appear to explain the increases in $\dot{V}O_{2max}$ and PO_{max}.

From an applied perspective, the decreased submaximal [Bla^-], $\dot{V}_E$, HR and P_{mask} suggest that a number of physiological stressors have been reduced with HOX, which could be of great benefit to firefighting. A reduction in $\dot{V}_E$ and [Bla^-] around V_{TN} with HOX also suggest that high intensity work could be achieved with a reduction in fatigue related to anaerobic metabolism. Alternatively, the maximum work tolerance was increased which could be of significant importance in the emergency response. This would make any occupation, where the wearing of a SCBA and protective clothing is mandatory, safer.

3.5 References

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CHAPTER 4

THE EFFECTS OF HYPEROXIA DURING SUBMAXIMAL EXERCISE WITH THE SELF-CONTAINED BREATHING APPARATUS

4.1 Introduction

For the last 75 years it has been generally accepted that hyperoxia (HOX) improves both maximal (Hughes et al. 1968; Ekblom et al. 1975; Byrnes et al. 1984; Peltonen et al. 1995; Nielsen et al. 1998) and submaximal performance (Wilson and Welch, 1975; Adams and Welch, 1980; Plet et al. 1992). These studies have generally addressed the effects of HOX on ventilatory, cardiovascular and metabolic parameters, as well as the possible mechanisms for the enhanced performance. However, relatively little research (Van den Berg et al. 1975; Petersen et al. 1999) has addressed the more applied question of how the ergogenic effects of HOX might be beneficial for everyday use. It is unlikely that HOX could be utilized practically during a sporting event due to the difficulty in constantly delivering the gas mixture. However in some occupations where the administration of HOX is relatively simple, the benefits could be substantial.

Firefighting is commonly acknowledged as a physically demanding and often highly dangerous occupation, that requires the physical ability to perform for long periods of time in extremely hostile conditions. Fully outfitted firefighters have an accentuated response to exercise (Lemon and Hermiston, 1977; Davis et al. 1982; White et al. 1989), which can be attributed to anxiety, increased body temperature from the protective clothing and environment. The occupation also presents another very interesting consideration to exercise physiologists. The self-contained breathing apparatus (SCBA) is one of the most important components of the personal protective equipment used by firefighters but it has its limitations. The weight (≈15.5 kg) and straps of the harness can greatly affect the mechanics of the chest wall which can ultimately affect ventilation (Louhevaara et al. 1985). In addition, the regulator used to supply the gas mixture increases the resistance to breathing on inspiration and the regulator valve system increases the resistance to breathing on expiration. Both of these factors will increase the work of breathing (W_b), especially with heavy exercise at high ventilations.

It is generally accepted that (Wilson and Welch, 1975; Ekblom et al. 1975; Byrnes and Mullin, 1981; Plet et al. 1992) that HOX decreases pulmonary ventilation ($\dot{V}_E$), which is likely due to the depressant effect of the increased P_aO_2 on chemoreceptor responsiveness (Wilson and Welch, 1975; Byrnes and Mullin, 1981; Plet et al. 1992). This reduction in $\dot{V}_E$ can have significant beneficial implications to firefighting and may alleviate some of the aforementioned limitations associated with the SCBA. Firstly, a decreased $\dot{V}_E$ will increase the life of the compressed gas tank, providing a greater reserve for emergency situations. Secondly, the reduction in $\dot{V}_E$ at submaximal levels with HOX, could reduce the work of breathing (W_b), associated with the restrictive properties of the SCBA and the increased resistance of the regulator and valves in the mask.

Two other possibly beneficial effects of HOX are the reduction in heart rate that has been reported at submaximal levels (Ekblom et al. 1975; Welch et al. 1977; Welch, 1982) and the reduced concentration of blood lactate ([Bla^-]) (Ekblom et al. 1975; Wilson et al. 1975; Welch et al. 1977 and Knight et al. 1996). Decreases in heart rate and [Bla^-] will reduce the stress placed on the cardiovascular and metabolic systems respectively.

Therefore, there appears to be a sound theoretical base for the hypothesis that HOX could have substantial benefits to the safety and performance of firefighters. However, the limited number of studies on the effects of HOX, in this unique population, have made fairly simple measurements of exercise performance and physiological stress (Van den berg et al. 1977; Petersen et al. 1999). Therefore, the aim of this study was to investigate the effects of HOX, during 20 min of submaximal treadmill exercise at V_T, while wearing firefighting equipment and breathing through the SCBA.

4.2 Methods

4.2.1 Subjects

Twenty-five moderately to well-trained non smoking males provided written informed consent to participate in the study which had previously received institutional ethics review board approval. Two subjects used in the study were asthmatic. However, they had a normal pulmonary function after the administration of a bronchodilator, which was used before every test. Twelve subjects were actively employed as firefighters and the remainder were University staff and students. In either case, each subject was completely familiar with breathing through an SCBA during vigorous exercise. Before entry into the study, subjects completed the revised Physical Activity Readiness Questionnaire (rPAR-Q) and subjects over 40 years old also completed a physician-supervised graded exercise test (GXT) with a 12-lead ECG. The physical characteristics (Mean $\pm$SD) of the subjects were as follows: age 32.8 ± 8.9 yr (range: 20 - 52); mass 85.86 ± 9.0 kg (range 67.5 - 100.5); height 179.3 ± 5.1cm (range:166 - 189); $\dot{V}O_{2max}$ 53.4 ± 6.6 $ml \cdot kg^{-1} \cdot min^{-1}$ (range: 45.1 - 68.9) and, maximum voluntary ventilation (MVV) $l \cdot min^{-1}$ ± (range)

4.2.2 Research Design

The experiment was conducted using a single blind, cross-over design which allowed investigation of the physiological responses to extended strenuous exercise while wearing firefighting gear and breathing through the SCBA. A graded exercise test with normoxic air (GXT_{21}), was administered to determine ventilatory threshold (V_T). The exercise intensity where V_T occurred was then used for the three experimental trials. Following GXT_{21} subjects completed a submaximal trial ($SUB_{21}P$) to 'practice' the experimental protocol at the prescribed intensity. Two further experimental trials (SUB_{40} (40.18%) and SUB_{21} (20.93%)) were then administered in a randomized fashion. At a convenient time prior to one of the tests listed above, resting pulmonary function measurements and MVV were taken using a computerized spirometer system (SensorMedics 2450). The two methods for gas administration were used as described in Chapter 3.

4.2.3 Graded Exercise Test (GXT)

V_T was determined by a walking test on a motorized treadmill, using a constant speed and gradually increasing grade protocol. After being weighed in just a pair of shorts, subjects dressed in National Fire Protection Association (NFPA) standard 1500 compliant protective clothing (mean weight SCBA = 21.8 ± .75 kg) as follows: pants; coat; helmet; flash hood; face piece and 1 glove; and a Scott 4.5 SCBA. Running shoes were used instead of firefighting boots for safety and comfort during the 20 minute trial. A single glove was worn to allow measurement of arterial saturation (S_aO_2) with a pulse oximeter (SensorMedics), and to maintain sterility at the blood sampling site.

Preceding the GXT subjects were given a brief warm-up, before the test speed was set at 3.5 m.p.h. With the speed constant, the treadmill belt was elevated at the rate of 2 % every two minutes until V_T was surpassed. V_T was detected by a systematic increase in the $\dot{V}_E/\dot{V}O_2$ ratio, while the ventilatory equivalent for $\dot{V}_E/\dot{V}CO_2$ remained constant or declined slightly. Wasserman (1987) has referred to this particular threshold as 'Stage II'. Pilot work had previously revealed that the exercise intensity associated with this 'threshold' represented a stressful, but tolerable load for extended (20 minute) exercise.

After V_T had been surpassed, the treadmill speed remained at the same brisk walking pace, while grade continued to be raised at the rate of 2% per minute until exhaustion. The highest 20 second reading for $\dot{V}O_2$ was accepted as $\dot{V}O_{2max}$ if at least two of the following criteria were met; a plateau in oxygen consumption (increases in $\dot{V}O_{2max} < 100 ml.min^{-1}$) were observed despite an increase in exercise load; a respiratory exchange ratio (RER)>1.15 was observed; age predicted or previously measured heart rate maximum was achieved; and/or, the subject was too fatigued to continue exercise.

Respiratory gases were collected using a rigid plastic cone, specially designed to fit over the exhalation ports of the SCBA regulator forming an air-tight seal. Expired gas from the breathing apparatus was monitored continuously and gas exchange variables were recorded every 20 seconds, with a SensorMedics 2900z metabolic measurement system (MMC) operating in mixing chamber mode.

4.2.4 Experimental Trials

The first experimental trial ($SUB_{21}P$) was designed to familiarize subjects with the 20 minute exercise protocol. A secondary function of $SUB_{21}P$ was to use the obtained results to assess the reliability of steady-rate exercise responses under normoxic conditions through a comparison with SUB_{21}. The two remaining trials (SUB_{21} and SUB_{40}) were administered in a random order with at least 24 hours between tests.

After a brief warm up, subjects walked for 20 minutes on a motor-driven treadmill at the intensity which elicited ventilatory threshold in GXT_{21} (VT_N). Throughout each trial, the subject was connected to the metabolic measurement system, (by the previously mentioned collection device). Heart rates were also continuously monitored using a Polar telemetry system.

During SUB_{21} and SUB_{40} blood samples were obtained from a MedexHi-Flo three way stopcock attached to an indwelling catheter in a dorsal forearm vein. Samples were taken prior to the warm up, at 5,10 and 15 minutes during the test and then again at the termination of the test (20 min). The catheter was kept patent by infusion of sterile saline after each sample was drawn. The SCBA was removed at the end of the 20 minute trials and the subjects followed a standardized cool down at 2.5 m.p.h on a level treadmill for 3 minutes and a final blood sample was taken at exactly 5 minutes post test. Immediately after sampling, 200μl of blood were transferred to a preservative tube (Yellow Springs Instruments, YSI 2315). The blood was then stored at -20°C until lactate analysis was performed. This process was accomplished within three weeks of the sample being taken. All samples were measured in duplicate with a YSI Model 27 analyzer.

For SUB_{40} and $SUB_{21,}$ pre and postest hematocrit (Hct) and Hemoglobin (Hb) were measured in duplicate using blood from the pre and 5 minute post exercise samples. The cyanmethemoglobin method (Stanbio laboratory inc) was used for the colorimetric determination of hemoglobin and samples were analysed using a spectrometer (Milton-Roy, Spectronic 601) set at a wavelength of 540nm. Hematocrit was measured using heparinized capillary tubes spun in a microhematocrit centrifuge for 5 minutes. These measurements were important for assessing the blood lactate concentrations and heart rate responses to exercise because of the significant sweat responses in the protective clothing. The effect on weight loss of this increased sweat output was also estimated during the 20 minute trials by getting subjects to remove the protective clothing, dry off all excessive moisture and re-weigh in shorts on the completion of each trial.

Inspiratory and expiratory mouth pressure was measured during SUB_{40} and SUB_{21} using a Validyne (DP45-28) differential pressure transducer. The transducer was connected to the face piece by a 5mm inside diameter hose, which was attached to the face piece by a specifically designed metal disc with a small metal nipple on the exterior surface. The disc replaced the cover of the voice port of the mask creating an air tight seal. The pressure transducer was connected to a Validyne (CD-19) amplifier which was subsequently attached to a heat sensitive chart recorder (General Scanning, RS6 5-P) . Inspiratory and expiratory mask pressure ($P_{mask)}$ was recorded for 15 seconds at 5,10,15 and 20 min

At five minute intervals throughout each trial subjects were asked to indicate a numerical rating from the fifteen point Borg Scale (Borg, 1982) which described how they felt 'overall' for the exercise they were performing. In conjunction with the rating of perceived exertion (RPE), perceptions of respiratory distress (PRD) were also measured using a 7 point psychophysical scale refined by Morgan and Raven (1985). Subjects were asked to indicate the rating which most closely matched their perception of breathing.

4.2.5 Statistical Analysis

All dependent variables, under the two experimental conditions, namely hyperoxia (40%) and normoxia (20.93%) and between the three experimental trials were analyzed using a repeated measures analysis of variance (ANOVA). The Scheffe post hoc multiple comparisons test, was used if appropriate. The alpha level was set at .05 for both the analysis of variance and the post hoc test.

4.3 Results

The reproducibility of measurements taken throughout the two normoxic experimental trials are summarized in Table 4-1. There were no significant differences in oxygen consumption ($\dot{V}O_2$), carbon dioxide produced ($\dot{V}CO_2$), respiratory exchange ratio (RER) or arterial saturation (S_aO_2) for measurements taken at 5 minute intervals throughout $SUB_{21}P$ and SUB_{21}. Likewise, there were also no significant differences in tidal volume (Tv), frequency of breathing (f) or the minute ventilation ($\dot{V}_E$). The only significant difference ($p<0.05$) found between the two normoxic trials was a lower heart rate after 15 minutes in the SUB_{21} trial. This lower heart rate observed in SUB_{21} was the only evidence of a learning effect. considered to represent a learning effect. Therefore, the results from SUB_{21} were used when assessing the treatment effect of SUB_{40}.

Table 4-2 gives a summary of the physiological responses observed throughout SUB_{21} and SUB_{40}. Mean values for measurements taken are reported at 5 minute intervals throughout the trials. The exercise intensities for SUB_{21} and SUB_{40} were not significantly different between the two trials (Figure 4.1) the mean power output was 150.3 ± (SD) 23.0W and 150.3 ± 23.3W under the normoxic and hyperoxic conditions respectively.

Oxygen consumption rose during both trials (Figure 4.2). This rise in $\dot{V}O_2$ resulting in a significantly higher oxygen consumption between the start and the end of the trial for SUB_{21} but not for SUB_{40}. However, no significant difference was found between treatments at any comparable time during the test. Carbon dioxide production also increased throughout the trials in conjunction with the increase in $\dot{V}O_2$. Again, no significant difference in $\dot{V}CO_2$ was found between SUB_{21} and SUB_{40} (Figure 4.3) There was also no significant difference in RER between the SUB_{21} and SUB_{40} at 10, 15 and 20 minutes. However, the initial 5 minute value was significantly lower in SUB_{40} (Table 2).

Ventilation during SUB_{40} was significantly lower ($p<0.05$) at all times throughout the trial (Figure 4.4). The suppression of $\dot{V}_E$ with 40% O_2 resulted predominantly from a significant decrease in mean tidal volume (T_V). However, toward the end of SUB_{40} the reduced T_V was also supported by a significantly lower respiratory rate (Table 4-2). The decreased $\dot{V}_E$ throughout SUB_{40} led to a significantly lower consumption of gas mixture needed to complete the trial (Figure 4.5) (1986.4 ± 34.65 l in SUB_{21} compared to 1771.0 ± 44.85 l in SUB_{40}).

The decrease in $\dot{V}_E$ throughout SUB_{40} was also accompanied by a significantly lower heart rate and a significantly higher S_aO_2 at all times during SUB_{40} (Figures 4.6 and 4.7).

Heart rate responses were parallel over the 20 minute trials. However, mean heart rate was 6-7 b·p·m lower with HOX.

Table 4-3 and Figure 4.8 show the P_{mask} findings throughout this study. P_{mask} on inspiration was not significantly different between SUB_{21} and SUB_{40}. Expiration values were significant lower at 5, 15 and 20 minutes during SUB_{40}. However, if these results are combined to obtain the pressure swing (P_{swing}) needed to complete one ventilatory cycle, the findings show a significant difference across all stages of SUB_{40} (Table 4-3). Additionally, when these finding are accompanied by the 10.3 to 14.4 l differences measured in $\dot{V}_E$, it can be assumed that the work of breathing (W_b) was also reduced.

Figure 4.9 shows that a markedly different response to blood lactate accumulation was observed during SUB_{40}. Resting [Bla^-] were the same, however after the start of exercise the differences in [Bla^-] were different ($p<0.05$) for the remaining 5 samples (Table 4-4). Mean [Bla^-] rose ($p<0.05$) from 2.86 $m·mol·l^{-1}$ at 5 minutes to 3.95 $m·mol·l^{-1}$ on completion of SUB_{21}. However, in SUB_{40} [Bla^-] was relatively constant, over the same time period and the 5 min recovery sample was not significantly different from rest. In contrast, [Bla^-] was still significantly elevated during the recovery period in the normoxic condition.

Table 4-5 summarizes findings for mass, Hct and [Hb] across SUB_{21} and SUB_{40}. A significant reduction in mass occurred over the two trials (0.85kg and 0.79kg in SUB_{21} and SUB_{40}, respectively). In conjunction with this finding, a significant increase ($p<0.05$) was observed in Hct and [Hb] over both trials. However, no difference in these variables was observed between trials.

Figures 4.10 and 4.11 depict the rating of perceived exertion (RPE) and perceived respiratory distress (PRD) throughout SUB_{21} and SUB_{40}. RPE was consistently lower throughout SUB_{40}. Values rose from a 5 minute reading of 9.7 to 12.6 in SUB_{40} and from 10.4 to 13.7 in SUB_{21}. These results demonstrate that the subjects perceived the exercise to be significantly harder from 10 minutes into the trial onwards ($p<0.05$). The PRD readings complement the ventilation results previously mentioned. PRD tended to be higher throughout SUB_{21} and was significantly higher by the end of the test. Although PRD rose within the SUB_{40} it should be mentioned that this rise was of a lesser magnitude than in SUB_{21}.

Table 4-1 : Reproducibility of selected physiological variables for the normoxic experimental trials (SUB$_{21}$P and SUB$_{21}$).

	SUB$_{21}$P			
	5	10	15	20
$\dot{V}O_2$	3.196 ± 0.07	3.252 ± 0.07	3.295 ± 0.07	3.331 ± 0.07
$\dot{V}CO_2$	3.045 ± 0.06	3.122 ± 0.07	3.168 ± 0.08	3.221 ± 0.07
RER	0.95 ± 0.01	0.96 ± 0.01	0.96 ± 0.01	0.97 ± 0.01
V_E	95.4 ± 1.97	104.2 ± 2.48	109.8 ± 2.97	117.0 ± 3.24
T_V	2.72 ± 0.09	2.80 ± 0.12	2.80 ± 0.12	2.86 ± 0.14
f	35.3 ± 0.93	38.1 ± 1.10	39.8 ± 1.18	41.9 ± 1.28
HR	157.5 ± 2.24	167.6 ± 2.31	174.1 ± 2.33	178.7 ± 2.28
SaO_2	93.5 ± 0.32	93.2 ± 0.37	93.1 ± 0.38	92.9 ± 0.38

	SUB$_{21}$			
	5	10	15	20
$\dot{V}O_2$	3.126 ± 0.07	3.233 ± 0.06	3.276 ± 0.07	3.342 ± 0.07
$\dot{V}CO_2$	2.977 ± 0.06	3.057 ± 0.07	3.118 ± 0.07	3.199 ± 0.07
RER	0.95 ± 0.1	0.95 ± 0.01	0.95 ± 0.01	0.96 ± 0.01
V_E	95.5 ± 2.17	101.7 ± 2.44	106.9 ± 2.68	112.9 ± 2.94
T_V	2.78 ± 0.10	2.80 ± 0.09	2.80 ± 0.10	2.83 ± 0.11
f	35.3 ± 1.08	36.6 ± 1.10	38.6 ± 1.27	40.7 ± 1.32
HR	155.1 ± 2.22	164.6 ± 2.31	170.6 ± 2.39*	175.5 ± 2.28*
SaO_2	93.5 ± 0.29	93.1 ± 0.32	92.8 ± 0.31	92.6 ± 0.33

Values are means ± SE for 25 subjects. Oxygen consumption ($\dot{V}O_2$) measured l·min^{-1} (STPD), carbon dioxide produced ($\dot{V}CO_2$) measured in l·min^{-1} (STPD), ventilation (V_E) measured in l·min^{-1} (BTPS), tidal volume (T_v) measured in litres, breathing frequency (f) measured in br·p·m, heart rate measured in b·p·m and arterial saturation (S_aO_2) measured as a percentage. * = significant difference between the two normoxic trials ($p<0.05$).

Table 4-2: Physiological and Psychophysical variables during the normoxic (SUB_{21}) and hyperoxic (SUB_{40}) experimental trials.

	SUB_{21}			
	5	10	15	20
$\dot{V}O_2$	3.126 ± 0.07	3.233 ± 0.06	3.276 ± 0.06	3.342 ± 0.07
$\dot{V}CO_2$	2.977 ± 0.06	3.057 ± 0.07	3.118 ± 0.07	3.199 ± 0.07
RER	0.95 ± 0.1	0.95 ± 0.01	0.95 ± 0.01	0.96 ± 0.01
V_E	95.5 ± 2.17	101.7 ± 2.44	106.9 ± 2.68	112.9 ± 2 .94
T_V	2.78 ± 0.10	2.80 ± 0.09	2.80 ± 0.10	2.83 ± 0.11
f	35.3 ± 1.08	36.6 ± 1.10	38.6 ± 1.27	40.7 ± 1.32
HR	155.1 ± 2.22	164.6 ± 2.31	170.6 ± 2.39	175.5 ± 2.28
SaO_2	93.5 ± 0.29	93.1 ± 0.32	92.8 ± 0.31	92.6 ± 0.33
RPE	10.4 ± 0.42	11.6 ± 0.42	12.8 ± 0.46	13.7 ± 0.53
PRD	1.36 ± 0.11	1.88 ± 0.16	2.40 ± 0.18	2.71 ± 0.20
	SUB_{40}			
	5	10	15	20
$\dot{V}O_2$	3.224 ± 0.06	3.285 ± 0.06	3.291 ± 0.07	3.301 ± 0.07
$\dot{V}CO_2$	2.965 ± 0.05	3.037 ± 0.06	3.089 ± 0.07	3.118 ± 0.07
RER	0.92 ± 0.01*	0.93 ± 0.01	0.94 ± 0.01	0.95 ± 0.01
V_E	85.2 ± 1.55*	90.9 ± 1.92*	94.4 ± 2.07*	98.5 ± 2.54*
T_V	2.62 ± 0.10*	2.62 ± 0.10*	2.69 ± 0.11*	2.66 ± 0.12*
f	33.5 ± 1.24	35.7 ± 1.30	36.9 ± 1.17*	38.2 ± 1.33*
HR	148.5 ± 2.26*	157.3 ± 2.37*	163.4 ± 2.36*	169.2 ± 2.52*
SaO_2	97.9 ± 0.17*	97.5 ± 0.14*	97.3 ± 0.15*	97.1 ± 0.19*
RPE	9.7 ± 0.38	10.9 ± 0.39*	11.8 ± 0.41*	12.6 ± 0.49*
PRD	1.24 ± 0.11	1.56 ± 0.14	1.88 ± 0.18*	2.04 ± 0.20*

Values are means ± SE for 25 subjects. Oxygen consumption ($\dot{V}O_2$) measured $l \cdot min^{-1}$ (STPD), carbon dioxide produced ($\dot{V}CO_2$) measured in $l \cdot min^{-1}$ (STPD), ventilation ($\dot{V}_E$) measured in $l \cdot min^{-1}$ (BTPS), tidal volume (T_v) measured in litres, breathing frequency (f) measured in br·p·m, heart rate measured in b·p·m, arterial saturation (S_aO_2) measured as a percentage, rating of perceived exertion (RPE) and rating of perceived respiratory stress (PRD). * = significant difference between the normoxic and hyperoxic trials ($p<0.05$)

Table 4-3 : Mask pressure values measured during the normoxic (SUB$_{21}$) and hyperoxic (SUB$_{40}$) experimental trials.

	5	10	15	20
SUB$_{21}$I	-3.69 ± 0.14	-3.80 ± 0.16	-4.29 ± 0.18	-4.52 ± 0.14
SUB$_{40}$I	-3.20 ± 0.50	-3.41 ± 0.15	3.55 ± 0.15	-3.81 ± 0.13
SUB$_{21}$E	3.85 ± 0.30	4.20 ± 0.32	4.71 ± 0.36	5.15 ± 0.34
SUB$_{40}$E	2.99 ± 0.17*	3.52 ± 0.22	3.66 ± 0.26*	4.13 ± 0.27*
SUB$_{21}$P$_{swing}$	7.55 ± 0.34	8.00 ± 0.40	9.00 ± 0.44	9.67 ± 0.37
SUB$_{40}$P$_{swing}$	6.19 ± 0.22*	6.92 ± 0.32*	7.22 ± 0.33*	7.94 ± 0.32*

Values are means ± SE for 18 subjects. Mouth pressure during inspiration in the two treatment conditions (SUB$_{21}$I and SUB$_{40}$I) measure in cmH$_2$O. Mouth pressure during expiration in the two treatment conditions (SUB$_{21}$E and SUB$_{40}$E) measure in cmH$_2$O. Pressure swing between inspiration and expiration in the two treatment conditions (SUB$_{21}$P$_{swing}$ and SUB$_{40}$P$_{swing}$) measure in cmH$_2$O.
* = $p<0.05$.

Table 4-4 : Blood lactate responses during the normoxic (SUB$_{21}$) and hyperoxic (SUB$_{40}$) experimental trials.

	Samples					
	1	2	3	4	5	6
SUB$_{21}$	1.22 ± 0.06	2.86 ± 0.12	3.30 ± 0.17	3.58 ± 0.19	3.95 ± 0.22	2.91 ± 0.19
SUB$_{40}$	1.25 ± 0.08	2.11 ± 0.11*	2.16 ± 0.13*	2.18 ± 0.13*	2.25 ± 0.14*	1.69 ± 0.12*

Values are means ± SE, n=23 (except for sample 1, n=22 and sample 6, n=24). Blood Lactate measured in m·mol·.l^{-1}. * = $p<0.05$

Table 4-5: Hematocrit and Hemoglobin changes before and after the normoxic (SUB_{21}) and hyperoxic (SUB_{40}) experimental trialsd.

	SUB_{21}	SUB_{40}
Bw_{Pre}	84.86 ± 1.80	84.97 ± 1.76
Bw_{Post}	84.01 ± 1.79*	84.18 ± 1.75*
Hct_{Pre}	44.29 ± 2.78	43.66 ± 2.32
Hct_{Post}	46.84 ± 0.64*	45.90 ± 0.51*
$[Hb]_{Pre}$	14.29 ± 0.35	14.15 ± 0.35
$[Hb]_{Post}$	15.63 ± 0.37*	15.29 ± 0.26*

Values are means ± SE for 23 subjects. Pre and post test body weight (Bw) measured in kilograms, Pre and post test hematocrit (Hct) measured as the ratio of cells:plasma and pre and post hemoglobin concentration ([Hb]) measured in $gm{\cdot}dl^{-1}$ * = significant difference between pre and post test measurements ($p<0.05$). † = significant difference between hyperoxic and normoxic conditions.

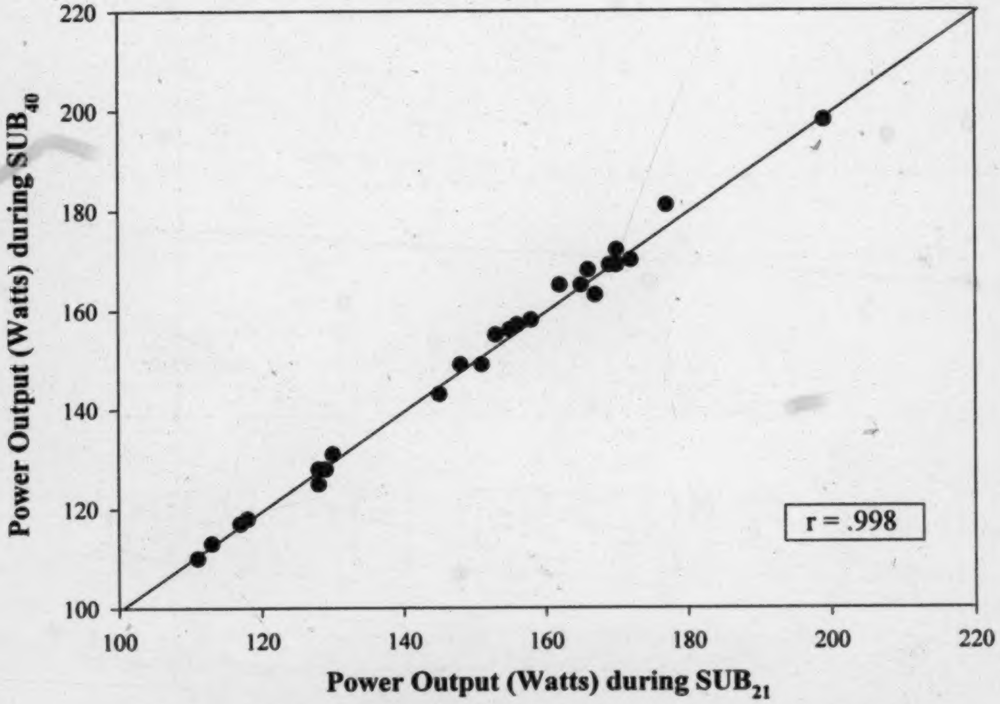


Figure 4.1 : Scatterplot (n=25) depicting the power output during SUB_{21} and SUB_{40}.

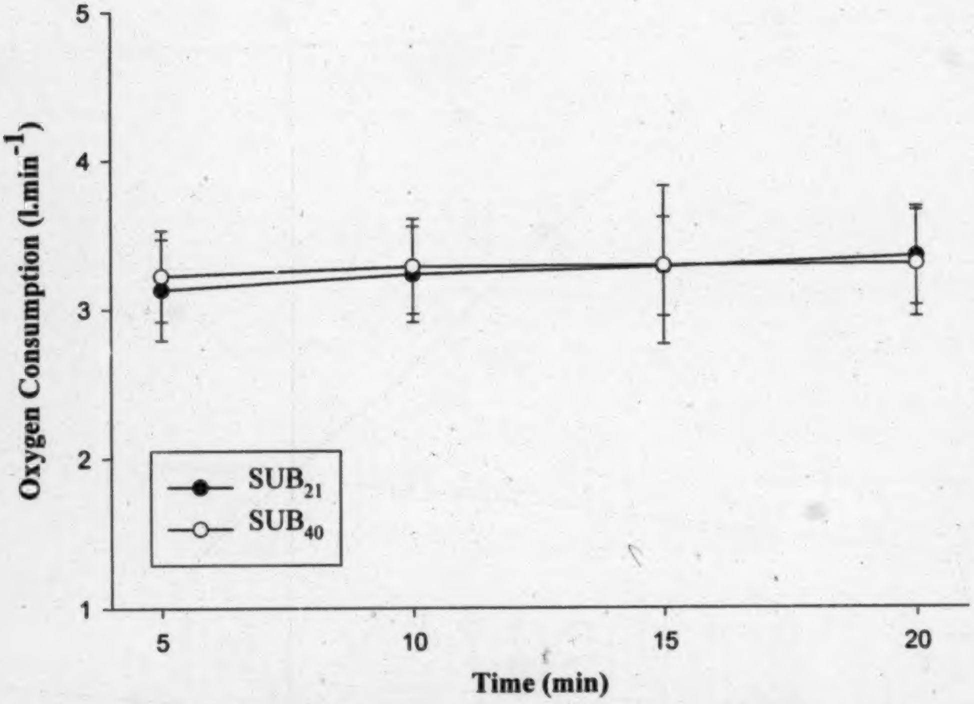


Figure 4.2 : Comparison of oxygen consumption measurements taken during SUB$_{21}$ and SUB$_{40}$. n=25.

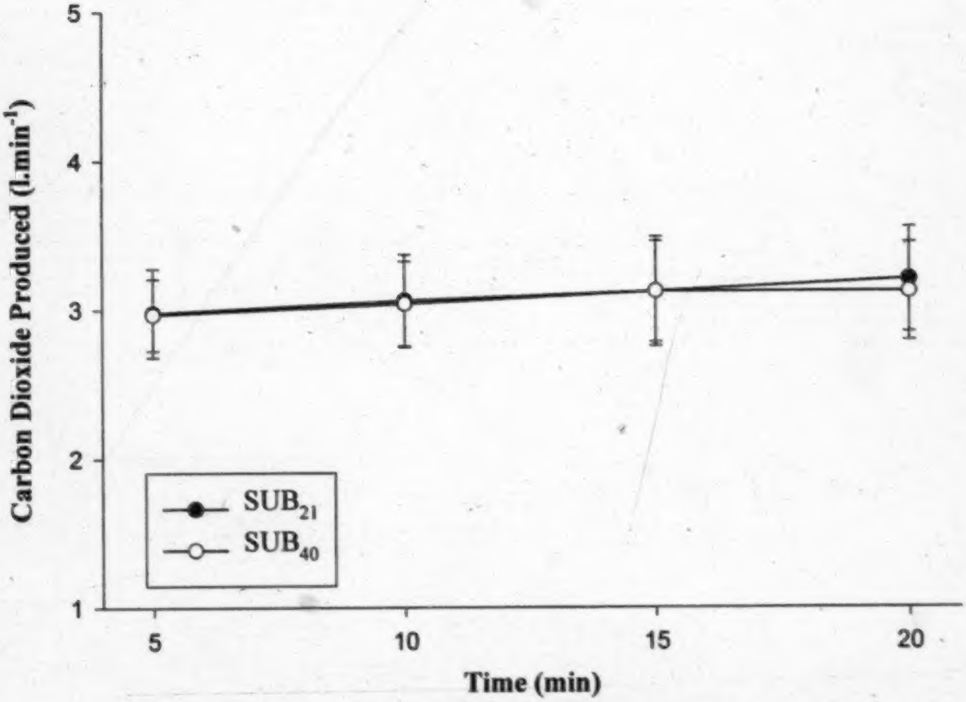


Figure 4.3 : Carbon dioxide produced during SUB$_{21}$ and SUB$_{40}$. n=25. *= $p<0.05$.

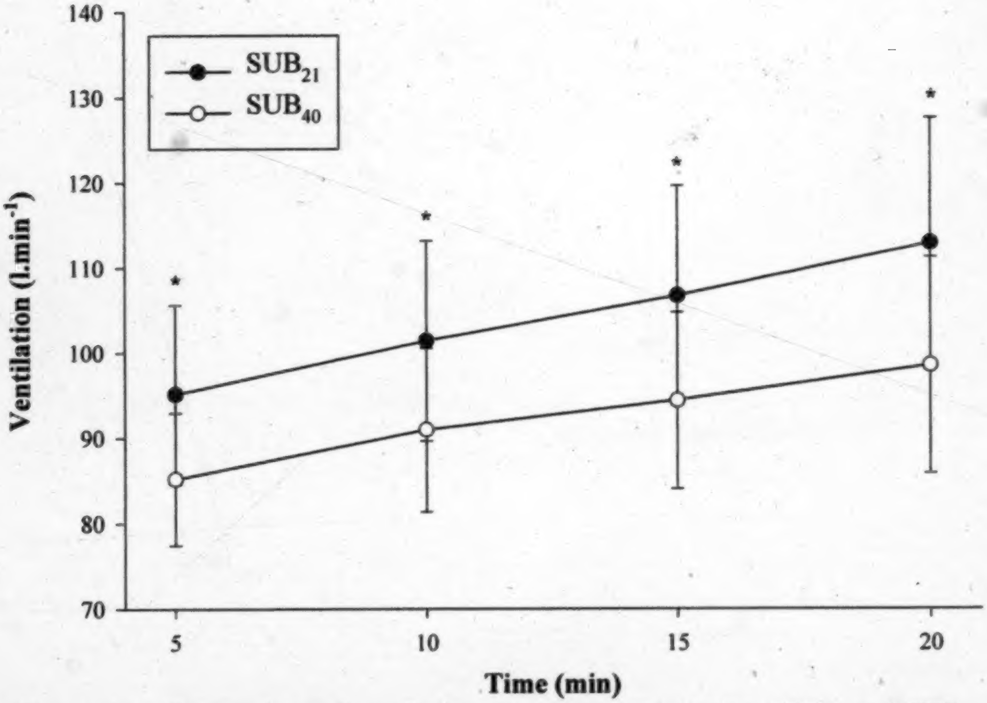


Figure 4.4 : Comparison of ventilation measurements during SUB$_{21}$ and SUB$_{40}$. n=25, * = $p<0.05$.

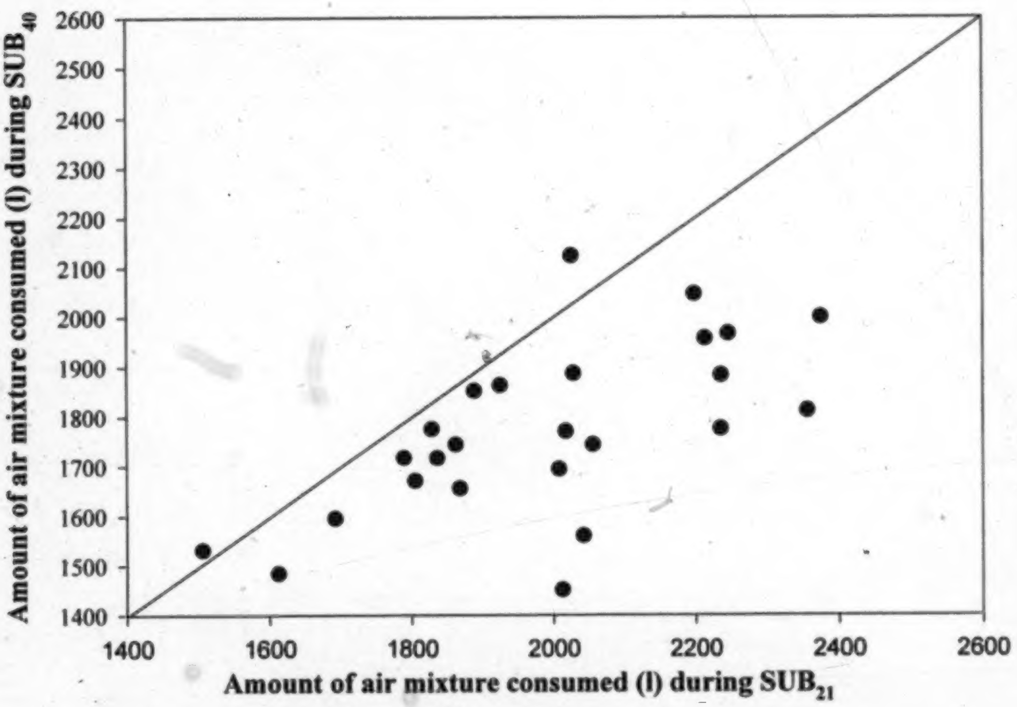


Figure 4.5 : A scatterplot (n=25) showing a the total amount of air mixture consumed during SUB_{21} and SUB_{40}.

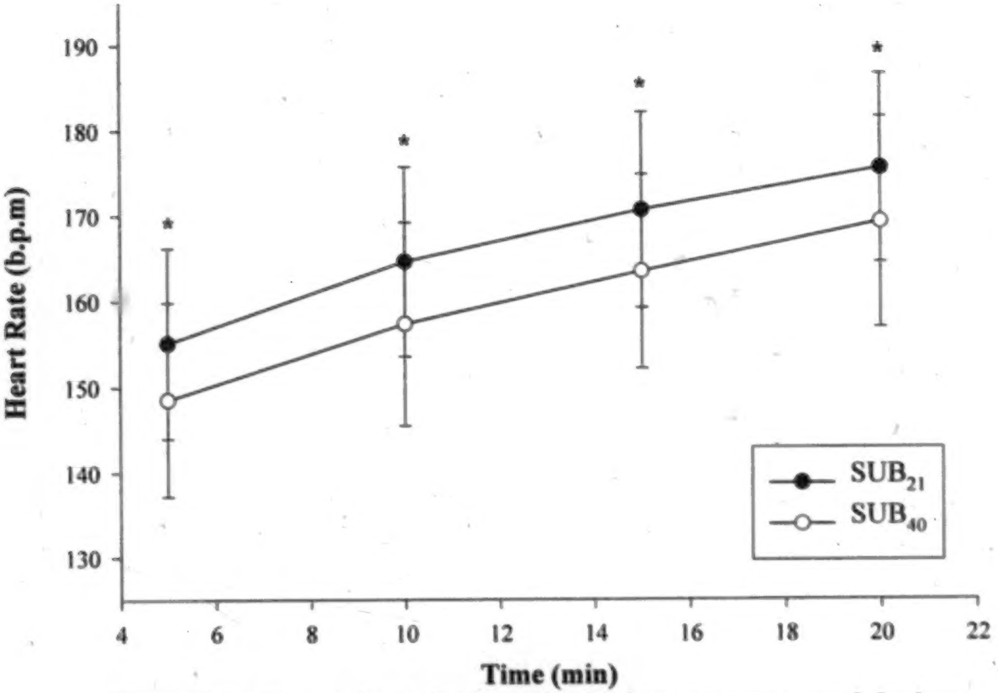


Figure 4.6 : Comparison of submaximal heart rates measured during SUB_{21} and SUB_{40}. n=25, * = $p<0.05$.

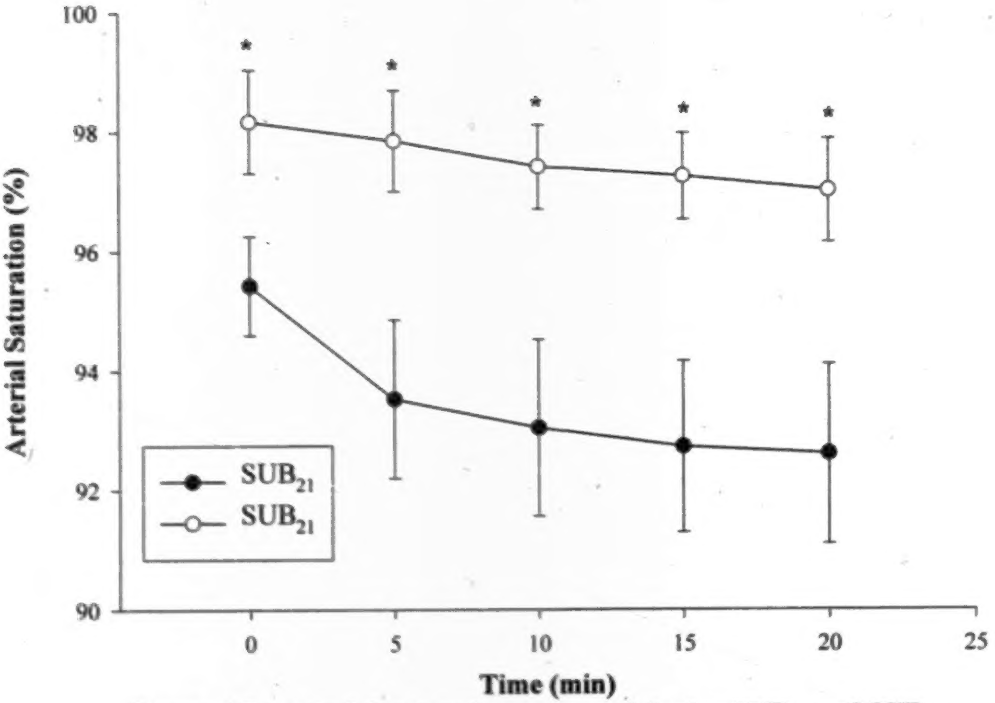


Figure 4.7 : Arterial saturation measured during SUB$_{21}$ and SUB$_{40}$.
n = 25. * = $p<0.05$.

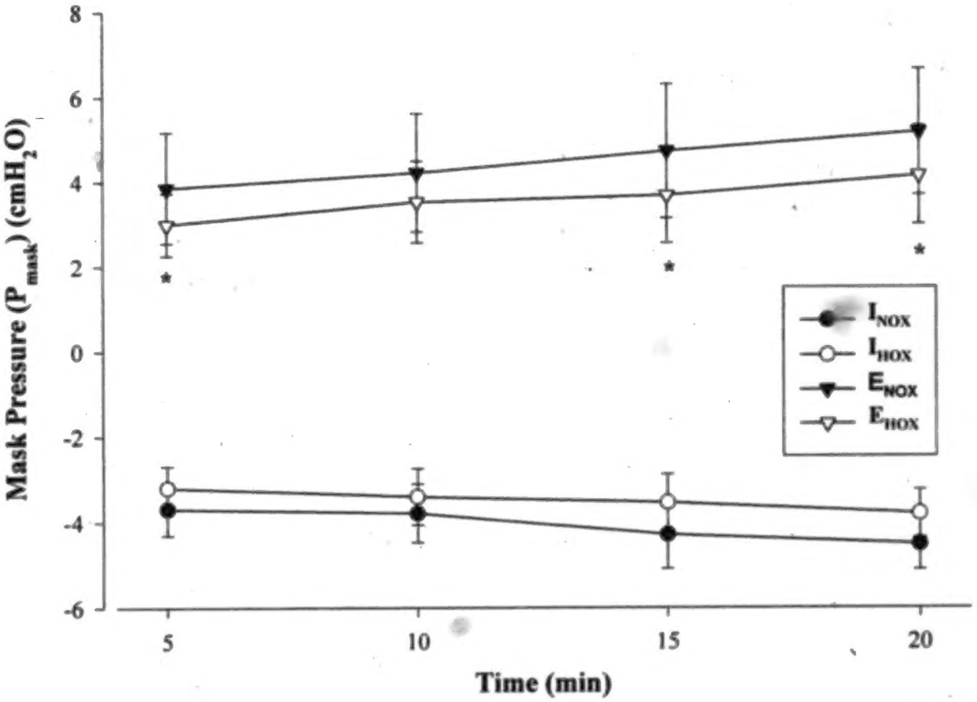
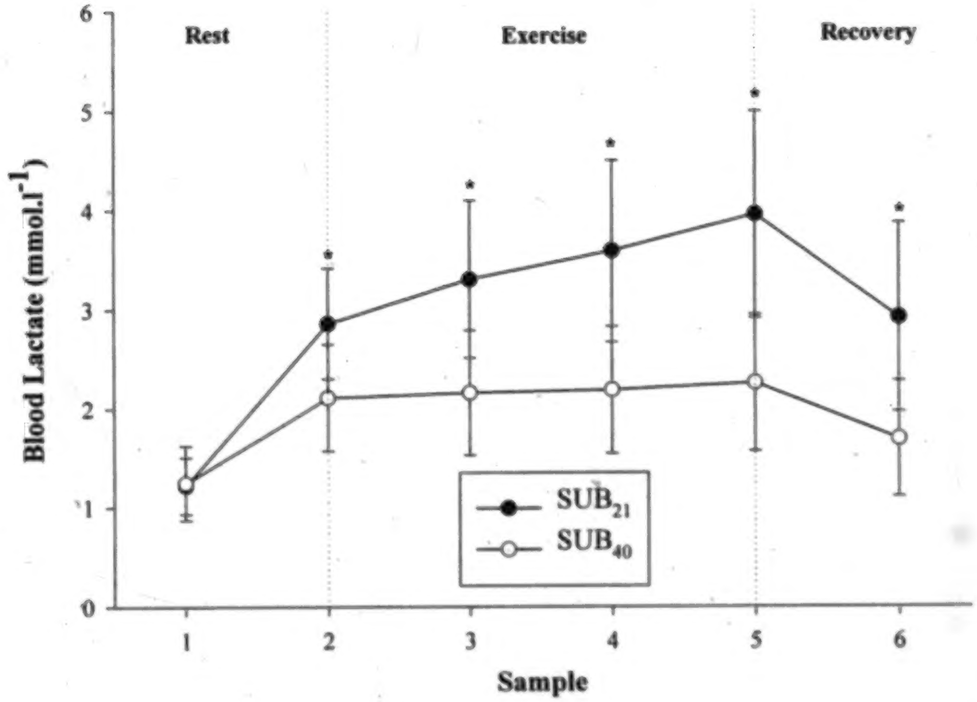


Figure 4.8 : Comparison of mask pressure on inspiration (I) and expiration (E) during SUB_{21} and SUB_{40}. n=18, * = $p<0.05$.



Graph 4.9 : Blood lactate responses during SUB$_{21}$ and SUB$_{40}$. n=22, * = $p<0.05$.

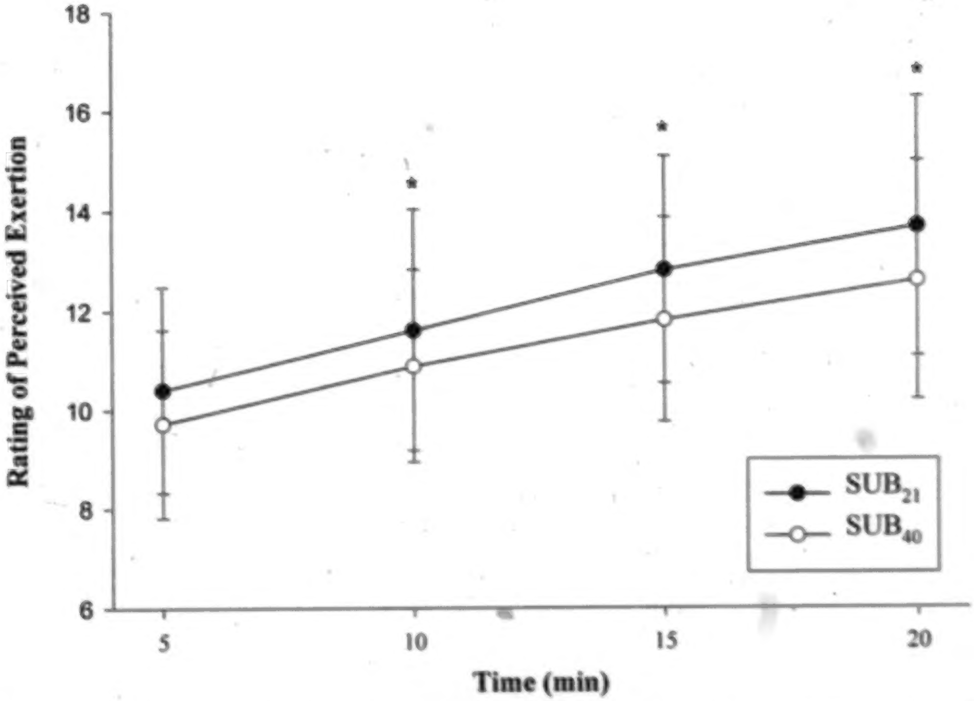


Figure 4.10 : Comparison of the Ratings of Perceived Exertion (RPE) measured during SUB$_{21}$ and SUB$_{40}$. n=25, * = $p<0.05$.

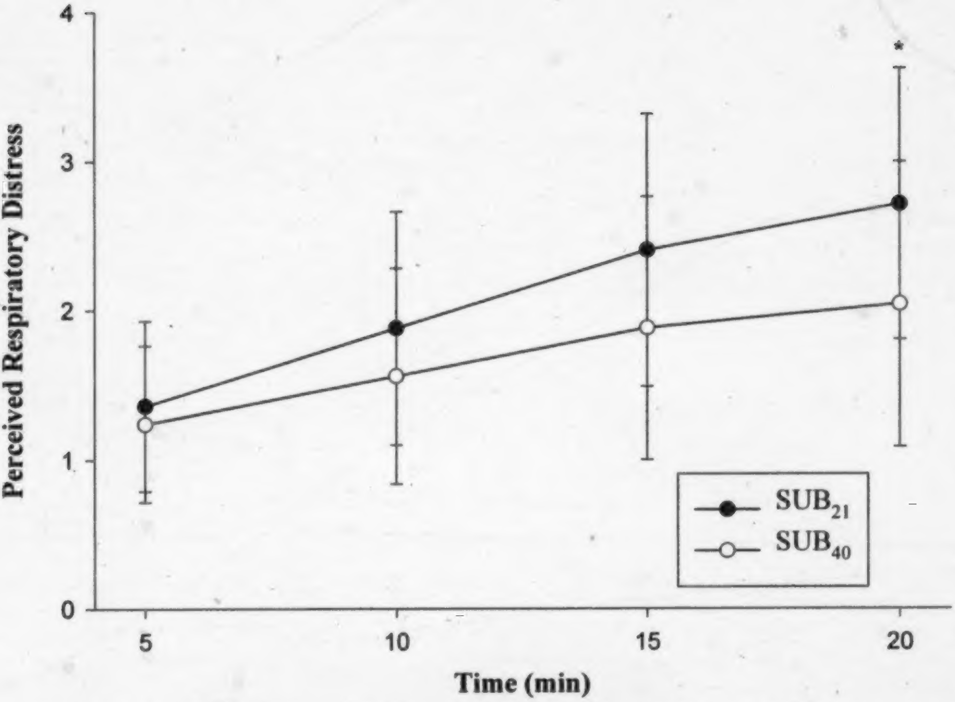


Figure 4.11 : Comparison of Perceived Respiratory Distress (PRD) measured during SUB_{21} and SUB_{40}. n=25, * = $p<0.05$.

4.4 Discussion

The results of this investigation showed that 40% O_2 significantly depressed $\dot{V}_E$ during the submaximal exercise protocol. The change in $\dot{V}_E$ coincided with a decrease in P_{mask} on both inspiration and expiration. These findings were accompanied by lower [Bla^-] and significantly lower heart rate throughout SUB_{40}. With respect to the use of the SCBA, the most relevant finding was the 10.8% decrease in the amount of gas mixture required to complete SUB_{40}.

The lower $\dot{V}_E$, apparently caused by the increased P_aO_2 with a subsequent suppression in the action of arterial chemoreceptors (Wilson and Welch 1975; Welch et al. 1974), is probably the most consistently reported finding within HOX research. We observed a 14.4 $l.min^{-1}$ reduction in $\dot{V}_E$ at the end of SUB_{40} which is consistent with previous findings (Hughes et al. 1968 (33% O_2); Ekblom et al. 1975 (50% O_2); Byrnes and Mullin, 1981 (70% O_2); Plet et al. 1992 (55% O_2); Miyamoto and Niizeki, 1995 (50% O_2)). In addition to the reduced $\dot{V}_E$ with SUB_{40}, expiratory P_{mask} was also significantly lower at 5, 15 and 20 minutes. Inspiratory P_{mask} also tended to be lower but no significant difference was found between SUB_{21} and SUB_{40} trials. The negative P_{mask} on inspiration was of lower magnitude than the positive P_{mask} on expiration because of the inspiratory pressure assist from the regulator.

The combination of the inspiratory and expiratory P_{mask} findings led to a significant reduction of 18.3% in the pressure swing generated across the ventilatory cycle. It seems feasible that this reduction in the swing of P_{mask}, combined with the reduction in $\dot{V}_E$ represents a reduction in the work of breathing W_b with HOX_{40}. This hypothesis could help explain the increased time to exhaustion that has been reported in the literature (Wilson and

Welch, 1975, Adams and Welch, 1980; Plet et al. 1991). A decrease in W_b should result in a decrease in the O_2 requirements of the respiratory muscles, which increases the O_2 availability for limb work (Harms et al, 1998). Shephard (1966) suggested that the upper limit for $\dot{V}_E$ during prolonged work is in the range of 120 $l.min^{-1}$. Above this critical value, the O_2 demands of the respiratory muscles can be elevated to an extent that the O_2 delivery to the working muscles is compromised (Bye et al. 1983). After 20 minutes of exercise during SUB_{21}, the $\dot{V}_E$ was 112 ± 2.94 $l.min^{-1}$, a value bordering on the useful limit for $\dot{V}_E$ suggested by Shephard.

From an applied perspective, any significant reduction in $\dot{V}_E$ during work with the SCBA could be advantageous, as long as P_aCO_2 does not increase to a level that could be detrimental. Our findings suggest that a substantial increase in the breathing time of an SCBA tank is achievable with a HOX_{40} gas mixture. From our pilot work, the estimated duration of the SCBA tank used in this study while breathing NOX would be approximately 30-35 minutes. However, in an actual firefighting situation where other factors such as environment and anxiety are involved, $\dot{V}_E$ could be further increased, which would decrease tank duration. It appears that a tank duration of about 25 minutes is a more realistic estimate (Personal Communication, Tim Gutterson), especially as firefighters want to leave the fire before the tank is completely exhausted. Taking this into consideration, if average tank life is 25 minutes during high intensity work, the 10.8% decrease in the amount of air mixture consumed would increase tank life by 2 min 42s. This would allow more time on task or alternately would provide a greater safety margin. These findings are supported by the previous work of Van den Berg et al. (1977) who reported that a reduction in $\dot{V}_E$ and an improved recovery time from high intensity intermittent work, led to a 6% decrease in the amount of gas mixture consumed with 40% O_2. The apparently greater reduction in the gas consumption in the present study can likely be explained by markedly different exercise protocols.

Another possible benefit of HOX for SCBA use is a possible protection against respiratory muscle fatigue. There is good support for the occurrence of respiratory muscle fatigue during submaximal exercise (Roussos and Macklem, 1977; Jardim et al. 1981; Pardy and Bye, 1985; Johnson et al. 1996). Tenney and Reese (1968) demonstrated that the maximum tolerable $\dot{V}_E$ that could be sustained for prolonged submaximal exercise was 55% of maximum voluntary ventilation (MVV). Results from the present study showed that from 15 minutes onwards during SUB_{21}, the ratio of minute ventilation to MVV ($\dot{V}_E$/MVV) exceeded 55%, reaching a peak of 58.6% at end-exercise. However, in SUB_{40} the $\dot{V}_E$/MVV ratio remained well below 55% during the trial, peaking at 51.1% at end-exercise. Freedman et al. (1970) and Tenney and Reese (1968) have suggested that a $\dot{V}_E$ of 50%-55% MVV could be sustained indefinitely. Therefore, it seems plausible that the reduction in $\dot{V}_E$ with HOX could delay the onset of respiratory muscle fatigue.

This hypothesis is supported by Bye et al. (1984), who reported the mean $\dot{V}_E$ of seven male subjects at the end of exercise while breathing air to be 122 $l \cdot min^{-1}$, which represented a $\dot{V}_E$/MVV of 67 ± 17%. In addition, a significant increase in time to exhaustion (at 80% of maximum work capacity) was reported (from 5.9 ± 1.3 min with air and 9.8 ± 4.8 min with HOX_{40}). Bye et al. (1984) also measured the occurrence of diaphramatic fatigue in their subjects by a decrease in the high frequency (150-350 Hz) to low frequency (20-46 Hz) power ratio (H/L). A reduction in this ratio indicates a reduction in high-frequency activity of the diaphrams myopotentials and/or an increase in amplitude of low frequency activity (Gross et al. 1979) and can be used to identify an inability of the diaphram to maintain a predetermined transdiaphragmatic pressure (Pdi). In Bye et al.'s study, 5 of the 7 subjects demonstrated diaphramatic fatigue on the basis of a 20% fall in the H/L ratio during exercise with air. Two subjects also displayed a decrease in end-expiratory Pdi. With HOX_{40}, there was a delay in the fall of H/L and no decrease in Pdi was observed This study demonstrated that respiratory muscle fatigue can occur during high-intensity short term exercise in air, but that the effects are delayed and/or reduced with HOX_{40}.

It is not known whether a sustained $\dot{V}_E$ of 55% MVV for 20 minutes would result in respiratory muscle fatigue in the moderate to highly fit subjects in the present study. However, it is important to note that the SCBA makes this study unique. Previous investigations have shown that the weight of the SCBA and the harness used to carry it effects gas exchange and hinders the natural oscillation of the thorax, particularly at higher ventilations (Louhevaara et al. 1985; Raven et al. 1975). The regulator used to deliver the gas mixture is designed to provide a pressure assist to aid inspiration at low ventilation rates. However, during heavy work, where pulmonary $\dot{V}_E$ can readily exceed 100 $l.min^{-1}$, the resistance to breathing is dramatically increased as demonstrated by the increased $_{Pmask}$ on inspiration and expiration. Therefore it seems possible that a $\dot{V}_E$/MVV ratio of 55% with the SCBA, could have significant impact on respiratory muscle work and may be too high for prolonged work when using a SCBA.

This is supported by the perceived respiratory stress (PRD) and S_aO_2 results from the exercise protocols. At the completion of SUB_{40}, the mean PRD had reached a value of 2.0 (2.04 ± 0.20) which corresponds to a subjective measure where breathing was still considered to be 'okay'. However, in SUB_{21}, mean PRD rose much closer to a reading of 3.0 (2.71 ± 0.20) which corresponds to a feeling of 'breathing hard'. The S_aO_2 data present a more tangible finding. Conceivably, if the respiratory muscles began to fatigue, there would be a significant drop in alveolar ventilation and in arterial saturation. Other research has shown this not to be the case (Boutellier and Piwko, 1992). However in this study, S_aO_2 values dropped significantly to a mean value of 92.6 ± 0.33 % suggesting that during heavy exercise with NOX_{21} and the SCBA, respiratory muscle fatigue could be a limiting factor to performance.

During SUB_{40}, improved work tolerance was reflected by significantly lower RPE values at 10, 15 and 20 minutes. As well, we observed significantly lower heart rates and blood lactate concentrations for similar levels of $\dot{V}O_2$. Decreased submaximal heart rate is commonly observed during exercise with hyperoxia. Our data show that the oxygen cost of exercise was the same for both conditions, and it seems unlikely that there should be any change in stroke volume between conditions. As noted above, the S_aO_2 values remained significantly higher during the SUB_{40} condition, and presumably a higher C_aO_2 permits the same level of oxygen transport with reduced cardiac output. The observation of decreased $[Bla^-]$ with 40% O_2 is in agreement with previous research (Linarsson et al. 1974; Welch et al. 1977; Gautier et al. 1978; Adams and Welch, 1980) and is of interest.

Reduced $[Bla^-]$ at a similar $\dot{V}O_2$ has led to the suggestion (Hogan et al. 1983) of either a substrate shift favoring lipid utilization and/or a subsequent glycolysis inhibition (Pasteur effect). This hypothesis has found support through the observation of constant or decreased $\dot{V}CO_2$ (Welch et al. 1974; Adams et al. 1986) during submaximal exercise with consistent or even slightly elevated $\dot{V}O_2$ (Hughes et al. 1968; Linnarsson et al. 1974; Wilson et al. 1975) leading to a lower respiratory exchange ratio (RER) (Hughes et al. 1968; Welch et al. 1974; Wilson et al. 1975; Adams et al. 1986). However with the exception of the 5 minute reading for RER during SUB_{40}, we found no significant differences for $\dot{V}O_2$, $\dot{V}CO_2$ and RER between the two conditions, which is in agreement with the work of Byrnes et al. (1984) and Miyamoto and Niizeki (1995).

Adams et al. (1986) and Byrnes and Mullin (1984) studied the substrate shift hypothesis using biochemical markers rather than metabolic parameters. Adams et al. postulated that if the increase in exercise tolerance was due to a substrate shift, then there should be a decrease in free fatty acid (FFA) stores and a subsequent increase in blood glycerol levels, without significant changes in either blood glucose or blood alanine concentrations. Their findings did not completely support this hypothesis; alanine levels did remain constant during 30 minutes of exercise at 75% $\dot{V}O_{2max}$, although no significant changes were found in FFA or glucose values and glycerol significantly decreased. Byrnes and Mullin (1981) also found no significant change in muscle glycogen utilization which does not support the concept of a substrate shift. However, they suggest that their results may be due methodological variability. The results of these two studies support the metabolic data from the present study suggesting that a substrate shift is unlikely. The findings from earlier studies could be explained by the methods used to measure $\dot{V}O_2$ and $\dot{V}CO_2$, considering that even slight measurement errors can lead to be substantial differences in RER.

Although the results of this study do not support an increase in FFA metabolism with HOX_{40}, we did note a significant reduction in the RER after 5 minutes of exercise. This may be explained by lack of a wash-in period before exercise. A wash-in period was excluded due to concerns over the duration of heavy exercise which could be sustained before having to change the SCBA tank . Pilot work suggested that even a five minute wash-in period increased the risk that a subject with high ventilation rates may not have enough gas to complete the trial. Secondly, since our aim was partially to simulate realistic occupational use of the SCBA , it was considered more appropriate to neglect the wash-in period than to shorten the trial. The lower RER at 5 min in SUB_{40} could result from the faster O_2 kinetics observed with SUB_{40} at exercise intensities above V_T (MacDonald et al. 1997). After a metabolic 'steady state' was reached, there was no further change in RER. In any event, our data do not support the concept of a significant shift in substrate utilization during exercise with HOX_{40}.

There was a similar, significant reduction in body mass following both SUB_{21} and SUB_{40} protocols. On average, the reduction was approximately1% of body mass for both conditions which was undoubtedly due to restricted thermoregulation with the protective clothing. Our subjects completed 20 minutes of submaximal exercise in a thermo-neutral environment, and one might speculate that the effects would be far more dramatic in actual fire suppression conditions. This finding is of interest and indicates that firefighters are at significant risk for dehydration.

The higher $[Bla^-]$ observed with SUB_{21} cannot be explained by a greater reduction in blood volume since there were no significant differences for change in body mass, Hct or [Hb] between SUB_{21} and SUB_{40},. The methods in this study cannot explain the differences in blood lactate between conditions, however three hypotheses may be considered. First, the increased W_b as indicated by the higher ventilation and increased P_{mask} during SUB_{21}, would logically lead to increased energy demand from the respiratory muscles. Given that the exercise intensity was at or near the anaerobic threshold, there could be a significant glycolytic contribution involved in meeting this demand. Second, during heavy exercise around or above V_T, redistribution of blood flow favoring the working muscles (Jardim et al 1981), may result in underperfusion of organs normally involved in lactate removal. If removal is compromised, the logical result would be and increase in [Bla^-]. Third, the higher exercise stress found with SUB_{21} may lead to an increase in blood catecholamine concentration, with a subsequent increase in [Bla^-].

Jardim et al. (1981) and Eldridge (1966) have shown that sustained breathing against an inspiratory resistance can lead to an increase in the amount of Bla^- produced by the respiratory muscles, with the exception of the diaphragm (Manohar and Hassan, 1990). Jardim et al. (1981) estimated that the resulting increase in [Bla^-] could be in the range of 1-3 $m.mol.l^{-1}$. This possibility alone would explain the mean 1.25 $m.mol.l^{-1}$ increase found in this study. It should be mentioned however, that the P_{mask} generated on inspiration in study by Jardim et al. were considerably higher than during the SUB_{21} protocol. While we cannot account for the effect of these factors, it should be remembered that the energy expenditure of the respiratory muscles could be further increased by the previously mentioned weight and restriction of the thorax from the SCBA and harness.

During the transition from moderate to high intensity exercise the amount of O_2 consumed by the respiratory muscles rises exponentially (Robertson et al. 1977) possibly reaching as high as 9 $ml\cdot l^{-1}\cdot V_E\cdot min^{-1}$ (Martin and Stager, 1981). The increase in the O_2 cost of breathing, without a decrease in blood flow to the working muscles would suggest that blood flow to other organs in the body must be compromised during heavy exercise with NOX_{21}. It therefore seems logical that if the O_2 delivery can be kept constant, either by an unchanged blood flow to the working muscles, or the higher C_aO_2 found with HOX (Ekblom et al. 1975; Horstman et al. 1976; Knight et al. 1993), then blood flow to other tissues may not decrease. There is some evidence that during submaximal exercise with HOX_{40}, blood flow to the working muscles is either unaffected (Knight et al. 1993) or even slightly reduced (Welch et al. 1977). Adams et al. (1986) suggest that an enhanced splanchnic blood flow with HOX_{40} may be responsible for their observations of decreased glycerol level. Subsequently, this argument could also be used for the decreased [Bla^-] found in SUB_{40}, as liver uptake of lactate would be proportional to the splanchnic flow.

During heavy exercise, increased secretion of adrenomedullary hormones enhances glycogenolysis (Richter et al. 1981 and Crowley et al. 1996), a finding is supported by the increased [Bla-] during treadmill exercise found in rats infused with epinephrine (Richter et al. 1981). These authors speculated that the increase in glycogenolysis is due to an increase in cAMP-dependent protein kinase activity, as phosphorylase b kinase is converted to the more active phosphorylase a kinase. Howley et al. (1983), found that during 40 min of cycle ergometer exercise at 67% of $\dot{V}O_{2max}$ with 30 minutes of 60% HOX breathing, followed by 10 minutes of NOX_{21} breathing, plasma ephinephrine concentrations were significantly reduced in comparison to when the gas mixtures were administered in a reversed fashion. This finding was in combination with a decrease in [Bla$^-$]. However, Howley et al. commented that even though this finding was significant, it was of a very small magnitude. It therefore seems possible that some of the elevated [Bla$^-$] with NOX$_{21}$ could be explained by increased catecholamines. However, the fact that RER values were similar suggest that any effect of the catecholamines is minor.

The findings of this study suggest that HOX$_{40}$ could improve the safety of any occupation that involves use of the SCBA. The decreases in submaximal V$_E$, and inspiratory and expiratory P$_{mask}$ suggest reduced ventilatory stress, which may be of more importance during prolonged work. Secondly, our results suggest that the reduction in ventilation found with HOX$_{40}$ will extend tank life by over 10%. This finding alone could dramatically improve the safety of the occupation, by allowing a greater reserve in the breathing supply. The improved tolerance to sustained heavy work as indicated by the reduced hearts rates, [Bla$^-$] and subjective measurements taken in this study show that a higher work intensity can be sustained at similar levels of fatigue, or alternately, any given submaximal intensity could be sustained with less fatigue. The findings of this study conclude that the use of HOX$_{40}$ could have numerous benefits in any occupation that requires high intensity work while breathing from a self contained breathing apparatus.

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CHAPTER 5

GENERAL DISCUSSION

The findings of this investigation support the hypotheses that were presented in Chapter 1. The use of 40% oxygen increases $\dot{V}O_{2max}$ and PO_{max} of subjects wearing firefighting equipment and the SCBA by the 10.1% and 9.3% increases, respectively. Secondly, HOX reduced heart rate after 5,10,15 and 20 min of strenuous constant rate exercise at V_{TN}. This was accompanied by decreases in $\dot{V}_E$, P_{mask} and $[Bla^-]$ which suggests that both ventilatory and metabolic stresses were reduced. Finally, HOX decreased the psychophysical stress as represented by the reduction in RPE and PRD during SUB_{40}. These findings support the literature that suggests HOX has an ergogenic effect on performance (Wilson et al.1975; Adams and Welch, 1980; Byrnes and Mullin, 1981; Plet et al. 1992; Peltonen et al. 1995; Knight et al. 1996;Nielsen et al. 1998), which could have a number of benefits to the applied setting of firefighting.

As previously mentioned in Chapters 1 and 2, the measurement of metabolic parameters under hyperoxic conditions have often been criticized (Welch and Pedersen; 1981; Welch 1982; Wagner, 1996). This is mainly due to the difficulty in avoiding room air contamination of the expired gas sample. However, throughout this study the recommendations of Welch and Pedersen (1981) were followed and extreme care was taken in the calibration of the metabolic cart used for the measurement of expired gas parameters (Appendix B). The findings suggest that efforts in this regard were successful. The subjects mass was very closely monitored and there was no significant difference between all tests. Consequently, the energy requirement at any workload should be the same for each subject, independent of the gas mixture. This hypothesis is supported by the finding of no difference between $\dot{V}O_2$ measurements up to and including 90% of $N\dot{V}O_{2\,max}$ in GXT_{21} and $GXT_{40.}$ Additionally, no significant difference in $\dot{V}O_2$ was measured at 5,10,15 or 20 min in SUB_{21} and SUB_{40}. Further support for the accuracy of our measurements can be gained from the amount of O_2 needed to generate 1W during the experimental trials. This was found to be 22.5 ± 2.5 ml/W and 22.1 ml/W ± 1.8 ml/W in SUB_{21} and SUB_{40} respectively. A similar finding 14.18 ± 0.8 ml/W and 14. 20 ± 1.1 ml/W, was also found at maximal exercise during GXT_{21} and $GXT_{40,}$ respectively, which supports the work of Peltonen et al. (1995). Therefore, it is with some confidence that we can postulate that the increase in power output found in GXT_{40} appears to be due to a rise in aerobic metabolism as no further contribution from anaerobic metabolism seems to have been made. This is supported by the similar $[Bla^-]$ at $\dot{V}O_{2max}$ in NOX and HOX.

The reason for the increase in $\dot{V}O_{2max}$ can only be hypothesized due to the limited number of measurements taken in this study that could help answer this question. However, many exercise scientists believe that there is a central limitation to $\dot{V}O_{2max}$ and if O_2 delivery can be increased, or kept stable by the use of O_2 fractions above 30% (Nielsen et al. 1998), then increases in $\dot{V}O_{2max}$ will occur. In this study, HOX was found to increase ($p<0.05$) and stabilize $S_aO_{2,}$ which would increase C_aO_2 and O_2 delivery to the same extent. Assuming no decrease in blood flow to the working muscles occurred as found by Knight et al. (1993 and 1996). The increase in $\dot{V}O_{2max}$ will be related to the improved O_2 delivery and an elevated a-vDO_2, depending on C_vO_2.

The effect of HOX on blood flow during exercise is discussed in Chapter 3. However, one further study should be mentioned (Roach et al. 1999). In this study Roach et al. used knee-extension ergometry to look at the extent that C_aO_2, independent of P_aO_2, would affect compensatory regulation when the body was challenged by hypoxemia. To achieve this, the authors looked at two conditions (hypoxia and anemia) with a matched C_aO_2 but markedly different P_aO_2 and two conditions (hypoxia and anemia + hypoxia) with matched P_aO_2 and different C_aO_2. The findings suggest that the regulation of cardiac output, leg blood flow and arterial O_2 delivery is dependent primarily on C_aO_2 or $[H_b]$, not P_aO_2 as previously thought. This would suggest that with HOX, the increased C_aO_2 reported (Ekblom et al. 1975; Welch et al. 1977; Knight et al. 1993), would cause vasoconstriction and therefore limit O_2 delivery, as suggested by Welch et al. (1977). This could explain why Nielsen et al. 1998, did not find any improvement in power output with their increases in $\dot{V}O_{2max}$. However, in this study, it seems feasible that the SCBA increases the O_2 cost of breathing, indicated by the increased P_{mask} and $\dot{V}_E$ at $N\dot{V}O_{2max}$ and during SUB_{21}. Therefore, at $N\dot{V}O_{2max}$ the respiratory muscles would demand more than the 14-16% of total cardiac output reported with normal mouth breathing (Harms et al. 1998). If this is the case, then the reduction in the W_b suggested by the reduced ($p<0.05$) $\dot{V}_E$ and P_{mask} at $N\dot{V}O_{2max}$, with HOX, may increase leg blood flow to an extent that the vasocontrictory effect of HOX is nullified. This would then allow O_2 delivery to increase in proportion to C_aO_2 which would seem to explain the approximately 10% increases in $\dot{V}O_{2max}$ and PO_{max} observed with HOX in this study.

Recent literature suggests that the respiratory system can limit maximal exercise, especially in highly trained athletes (Dempsey et al. 1984; Powers et al. 1984; Williams et al. 1986; Powers and Williams, 1987). However, it appears from this study and the previous work of Lohevaara et al. (1985) and Raven et al. (1977), that the SCBA could also effect maximal and submaximal performance in 'normal' subjects. It appears possible that the mechanical constraint to the thorax with the SCBA and the increased resistance to breathing from the regulator and expiration valves, could have a significant effect on the pulmonary system. In combination with an accentuated Bohr effect, these factors may be responsible for the arterial desaturation observed in GXT_{21} and SUB_{21}. A similar mechanical constraint was observed by Dempsey et al. (1984), who found that the tidal flow at $\dot{V}O_{2max}$ exceeded the limits of the resting maximal flow-volume curve, in highly trained athletes. However, when He-O_2 breathing (21% O_2 : 79% He) was used to 'mechanically unload' the respiratory muscles, the tidal breath's flow-volume loop at $\dot{V}O_{2max}$ returned to within the limits of the maximum flow-volume loop.

The decrease in W_b observed with He-O_2 by Dempsey et al. (1984) could be greatly beneficial to firefighting. He-O_2 has a viscosity 1.1 times that of air but a density of only 0.34 times that of air (Murphy et al. 1969). Thus, flow should remain laminar at higher intensities with He-O_2, reducing turbulence and airway resistance for a given muscular effort. This would result in a reduced W_b and could improve performance, as demonstrated by the higher $\dot{V}O_{2max}$ scores found by Brice and Welch (1983), Powers et al. (1986) and Esposito and Ferretti (1997). Secondly, He-O_2 could substantially reduce the elevated core temperatures associated with exercise in the protective clothing, as helium has a much greater thermal conductivity than nitrogen (Fox et al. 1966). However, one of the main findings of this study, in relation to the prolonged submaximal exercise, was the 10.8% increase in tank life with HOX, due to the suppression of $\dot{V}_E$. Conversely, He-O_2 has been shown to increase $\dot{V}_E$ (Wilson and Welch, 1980; Brice and Welch, 1983; Krishnan et al. 1997) which could have a detrimental effect to the duration of time that a working firefighter could breathe from a single tank. It appears that this possibly negative effect of He-O_2, could be at least partially corrected by the use of a He-HOX mix (40% O_2 : 60% He). Wilson and Welch (1980) demonstrated, that when a He-HOX mix (80% O_2 : 20% He) was delivered to ten male subjects during treadmill running, maximal $\dot{V}_E$ was not significantly different from either 20% O_2 : 80% N_2 or 80% O_2 : 20% N_2 breathing. This would suggest that although we might not be able to increase the life of a tank with He-O_2, the possible benefits from a decreased work of breathing and a reduced thermal stress could be substantial and it warrants further research.

Throughout the SUB_{40} trial, the significantly lower and more stable [Bla^-] and $\dot{V}_E$, while working at V_{TN} intensity, suggests that the thresholds for ventilation and lactate may have been displaced to a higher absolute power output with SUB_{40}. Unfortunately, no conclusion can be drawn from our findings since the study was not designed to investigate the effect of HOX on anaerobic threshold (A_T). The protocol used during the GXT's was designed to assess the maximal responses to HOX in full firefighting gear and the SCBA and the move to workloads of minute duration, shortly after V_T makes it difficult to accurately assess whether a 'shift' in V_T occurred. The reduced [Bla^-] and $\dot{V}_E$ at the workload that elicited V_{TN} and at the workload immediately afterward, does suggest that a displacement of V_T and L_T to a higher workload, may have occurred. This would agree with the findings of Miyamoto and Niizeki (1995) who found that 50% O_2 delayed the onset of isocapnic buffering until a much higher workload had been obtained. However, it should also be mentioned that Sadowsky et al. (1995) found that 40% hyperoxia failed to alter L_T during a graded cycle test.

The lactate and ventilatory thresholds are of particular importance when applied to the occupation of firefighting. L_T and V_T are estimations of the highest level of exercise that can be maintained for prolonged periods. Therefore, the intensity at which a firefighter can work without the detrimental effects of the anaerobic metabolism is related to the work level at which V_T and L_T occur. If HOX could elevate this intensity, then firefighters could achieve the same amount of work but with a significantly reduced ventilatory and metabolic stress. Alternatively, firefighters could also work at higher intensities without exceeding L_T and V_T. Both of these possibilities could substantially improve job safety. This question should be researched further. A slightly altered protocol which allowed for longer work periods and more frequent blood sampling would allow assessment of metabolic and ventilatory parameters with HOX while wearing the full protective clothing and SCBA and could further support the potential benefits of HOX to firefighting.

In conclusion, the objective of this investigation was to study the ventilatory, metabolic and cardiovascular responses to HOX, during both maximal and submaximal exercise, in subjects wearing full protective clothing and the SCBA. The results found that HOX increased $\dot{V}O_{2max}$ and PO_{max} and reduced HR, $\dot{V}_E$, P_{mask}, [Bla^-] and PRD during a prolonged submaximal trial at an intensity at or above V_{TN}. These findings demonstrate that HOX can improve work tolerance, a finding which could be of considerable benefit to firefighting. Additionally, the reduction in $\dot{V}_E$, P_{mask} and PRD during SUB_{40} appear to indicate that HOX can decrease the W_b and would imply a reduction in ventilatory stress. Likewise the reduction in [Bla$^-$] observed at submaximal levels supports the hypothesis that HOX can also reduce metabolic stress.

It can therefore be concluded, that 40% O_2 could have a beneficial effect on the performance and safety of firefighters. A decrease in ventilatory and metabolic stress could help reduce the level of fatigue that firefighters have to work under and the reduced $\dot{V}_E$ could increase the duration of an SCBA tank by approximately 10%. These findings could be of considerable significance if the life of a firefighter or the people that s/he is trying to rescue, depended on it.

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APPENDIX A

RELIABILITY BETWEEN THE TWO METHODS OF GAS DELIVERY

Introduction

As mentioned in chapters 3 and 4, two different methods were used to deliver the inspired gas mixtures. Method 1 was used with 10 subjects at the beginning of the experiment, but proved to be logistically difficult. Method 2 permitted several tests to be completed from one cylinder of either compressed air or the hyperoxic gas mixture and was used with the remaining 15 subjects. The reliability of the two methods was evaluated with the data from five subjects who each participated in two additional experimental trials, with the subjects walking for 20 minutes at V_T. The order of these tests was randomized and the reliability between the two methods calculated

Methods

Method 1 : The first 10 subjects used normoxic (20.93%) and hyperoxic (40%) mixtures from identical Scott 4.5 self-contained breathing apparatus (Figure C-1). Gas mixtures were delivered from the SCBA tank carried on the subject's back through a hose coupling, a pressure reducer, and a modified regulator (with the air saver switch, and Vibralert warning devices inactivated).

Method 2 : The remaining 15 subjects had the gas mixtures supplied from two K - size cylinders located adjacent to the treadmill (Figure C-2). The tanks and related fittings were covered so that the subjects could not identify the source of the gas mixture. The gas was supplied to the subject with the same hose, pressure reducer and regulator as described above. The subject carried a half full Scott tank to account for the weight and restriction normally associated with the SCBA and harness. A second pressure reducer was also attached to the harness, since the weight of the one on the breathing line was supported by the handrail of the treadmill

Results

The results of this procedure revealed that there was no difference in any of the selected physiological ($\dot{V}_E$, $\dot{V}O_2$, HR, S_aO_2,) or psychophysical responses (PRD, or RPE) to prolonged submaximal exercise at V_T between the two methods (Table A-1).

Table A : Comparison of physiological and psychophysical responses to prolonged exercise using the two different methods of delivery the gas mixtures.

	5	10	15	20
$\dot{V}O_2$ (Method 1)	2.900 ± .16	2.979 ± .12	3.061 ± .15	3.026 ± .15
$\dot{V}O_2$ (Method 2)	2.890 ± .15	2.956 ± .15	3.010 ± .16	3.016 ± .15
$\dot{V}_E$ (Method 1)	85.3 ± 7.5	92.1 ± 6.2	99.9 ± 7.3	104.3 ± 6.7
$\dot{V}_E$ (Method 2)	86.9 ± 5.7	93.5 ± 5.5	99.6 ± 5.8	107.5 ± 4.53
HR (Method 1)	160 ± 2.8	167 ± 2.7	173 ± 3.3	178 ± 3.8
HR (Method 2)	160 ± 2.5	172 ± 1.7	178 ± 2.5	181 ± 2.5
S_aO_2 (Method 1)	93.5 ± .29	92.5 ± .65	92.8 ± .48	93.0 ± .82
S_aO_2 (Method 2)	93.8 ±.25	92.5 ±.29	92.8 ± .25	92.5 ± .29
PRD (Method 1)	1.4 ± .3	2.0 ± .5	2.4 ± .5	3.0 ± .6
PRD (Method 2)	1.4 ± .3	2.0 ±.5	2.8 ± .5	3.0 ± .6
RPE (Method 1)	10.8 ± .8	12.0 ± .7	13.0 ±.6	13.8 ± .6
RPE (Method 2)	11.0 ± .7	12.4 ± .8	13.2 ± .5	14.2 ± .7

Values are means ± SE for 5 subjects. Oxygen consumption (VO_2) measured $l.min^{-1}$ (STPD), ventilation (V_E) measured in $l{\cdot}min^{-1}$ (BTPS), heart rate measured in $beats{\cdot}min^{-1}$, arterial saturation (SaO_2) measured as a percentage, rating of perceived exertion (RPE) and rating of perceived respiratory stress (PRD).

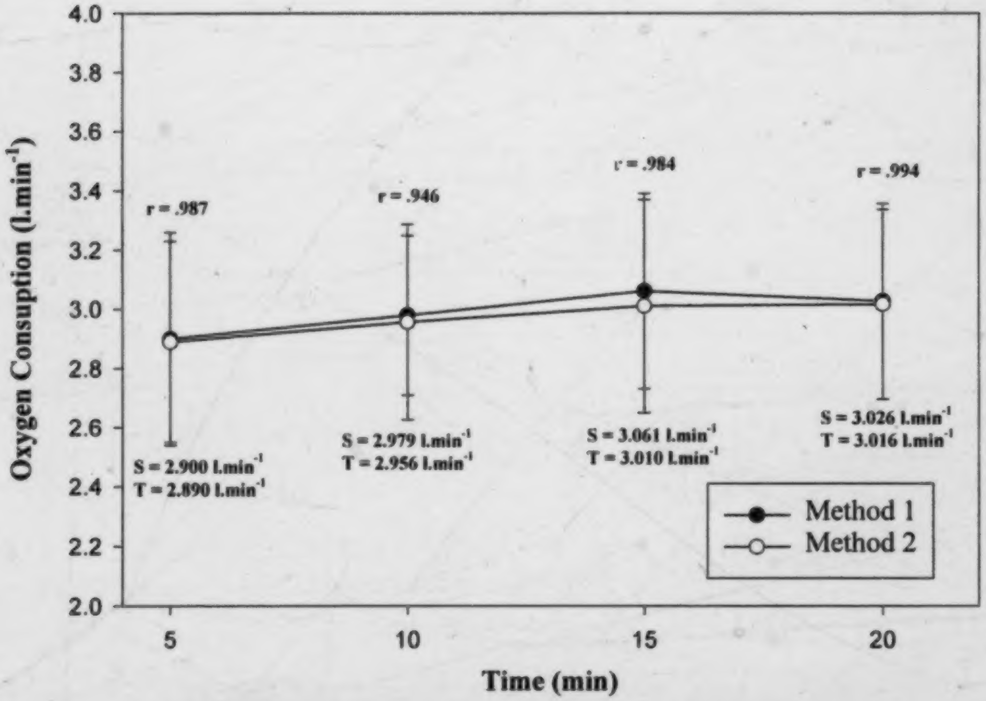


Figure A-1: The reliability of oxygen consumption measures during a 20 minute submaximal trial at V_T, using the two different methods for delivering inspired air (n=5). * = p<0.05.

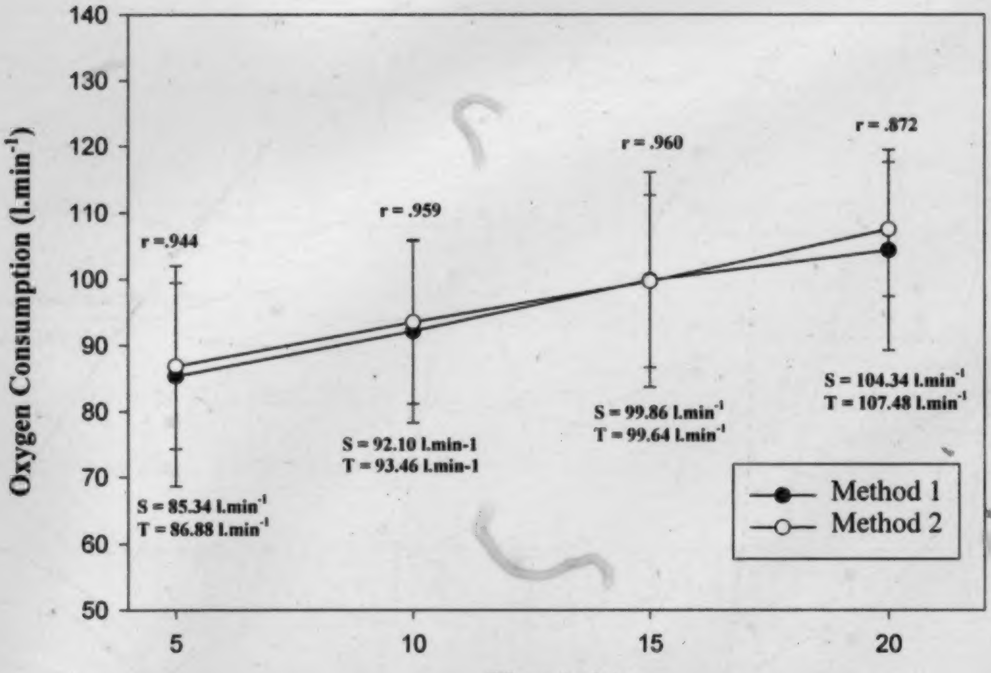


Figure A-2: The reliability of ventilation measures during a 20 minute submaximal trial at V_T, using the two methods for delivering inspired air (n=5). * = $p<0.05$.

APPENDIX B

ADDITIONAL METHODOLOGY

B.1 Graded Exercise Tests ($GXT_{21}P$, GXT_{21} and GXT_{40})

1. All graded exercise tests $GXT_{21}P$, GXT_{21} and GXT_{40} were administered in identical fashion to ensure subjects could not determine between the different tests.
2. $GXT_{21}P$, and GXT_{21} were administered as the first and second tests to all 25 subjects. Subjects were unaware that the 6 tests were not completely randomly. However, GXT_{40} was randomized with the two experimental trials SUB_{21} and SUB_{40}
3. On entering the lab subjects were weighed in shorts and T-shirt.
4. Subjects dressed in National Fire Protection Association (NFPA) standard 1500 compliant protective clothing as follows: pants; helmet; flash hood; 1 glove; and a Scott 4.5 SCBA including the face piece. An appropriate size face piece was selected and an air tight seal was created by pulling the mask very tight. This ensured that there was no leakage or contamination of the expired gas, even at the very high ventilations associated with $\dot{V}O_{2max}$.
5. Subjects were weighed again to ensure that an power output could be accurately calculated. This was necessary because there was slight individual difference in the weight of the clothing and SCBA.
6. Subjects were given an adequate warm up, which normally consisted of 5 minutes of walking on a level treadmill. The treadmill was then stopped and subjects were encouraged to stretch.
7. During the warm up, a SensorMedics 2900z Metabolic Measurements System (MMC) was calibrated. This process was repeated shortly before the test commenced. Immediately preceding the test, a sample of the F_IO_2 being

administered was sampled by the MMC out of the view of the subject (Figure C-4).

8. Respiratory gases were collected using a rigid plastic cone which had been specially designed to fit over the exhalation ports of the SCBA regulator forming an air-tight seal (Figure C-3).

9. The distal end of the cone was linked to the metabolic measurement system using a plastic gas collection hose.

10. Expired gases from the breathing apparatus were collected continuously and gas exchange variables were recorded every 20 s by the MMC operating in mixing chamber mode.

11. Inspiratory and expiratory mask pressure was measured in all subjects throughout the GXT. The pressures were quantified with a Validyne differential pressure transducer (DP45-28). The transducer was connected to the face piece by a 5 mm inside diameter hose attached to a specifically designed metal disc with a small metal nipple on the exterior surface. The disc replaced the cover of the voice port of the mask, creating an air tight seal. The pressure transducer was connected to a Validyne (CD19) amplifier which was subsequently attached to a chart recorder (General Scanning, RS6 5-P). Inspiratory and expiratory mask pressure was recorded for the last 15 seconds of every work load, and at maximal exercise for the GXT.

12. Immediately preceding the start of the test the pulse oximeter was attached to the subject's finger and the modified regulator and tube for measuring P_{mask} were attached to the subject's face piece

13. Treadmill speed was then increased to 3.5 m.p.h and elevation was raised to a 2%

grade and the test was started.

14. The speed was kept constant at 3.5 m.p.h for the entire test. Grade was raised 2% every 2 minutes until V_T had been surpassed. Following the occurrence of V_T the grade was raised 2% every minute until volitional exhaustion.

15. V_T was detected by a systematic increase in the $\dot{V}_E/\dot{V}O_2$ ratio, while the ventilatory equivalent for $\dot{V}_E/\dot{V}CO_2$ remained constant or declined slightly.

16. The highest 20 second reading for $\dot{V}O_2$ was accepted as $\dot{V}O_{2max}$ if at least two of the following criteria were met; a plateau in oxygen consumption (an increase in $\dot{V}O_2 <$ 100ml.min-1) was observed despite an increase in exercise load; a respiratory exchange ratio (RER) > 1.15 was observed; age predicted or previously measured heart rate maximum was achieved; and /or, the subject was too fatigued to continue exercise.

17. Heart rate was continuously monitored throughout each GXT using a Polar telemetry system.

18. Four blood samples were taken during each GXT (For more detailed see B.3)

19. Throughout the GXT the Rating of Perceived Exertion (RPE) and Perceived Respiratory distress (PRD) were recorded, on completion of each workload.

20. Toward the end of the test, when subjects were becoming fatigued and elevations were fairly steep, a spotter was employed behind the subject to ensure their safety.

21. Immediately on completion of each GXT the subject's SCBA, face piece, helmet, flash hood and glove were removed and subjects completed a standardized cool down. Which involved walking on a level treadmill at 2.5 m.p.h for three minutes.

B.2 Experimental Trials ($SUB_{21}P$, SUB_{21} and SUB_{40})

1. The first experimental trial ($SUB_{21}P$) was administered during the first week of testing after $GXT_{21}P$ and GXT_{21}.
2. The two remaining trials (SUB_{21} and SUB_{40}) were administered in a random order with GXT_{40} during the second week of testing.
3. Preceding each trial, subjects were weighed in only running shorts, and then again when wearing the full bunker gear and SCBA.
4. During and adequate warm up of walking and stretching, the MMC was calibrated. This process was then repeated shortly before the test commenced.
5. Immediately preceding the test, a sample of the F_IO_2 was sampled by the MMC out of the view of the subject (Figure C-4).
6. On successful calibration of the MMC, the subject's regulator, pulse oximeter and tube for measuring P_{mask} were connected, and the test was begun.
7. The subject then walked for 20 minutes on a motor-driven treadmill at the intensity which elicited VT_N.
8. Throughout each trial, the subject was connected to the MMC, (by the previously mentioned collection device) which allowed the measurement of expired metabolic parameters.
9. Inspiratory and expiratory P_{mask} were measured as explained for the GXT testing above.
10. Heart rate were also continuously monitored using a Polar telemetry system.
11. Six blood samples were taken during each experimental trial (For more detailed see

B.3)

12. Throughout the 20 minute trials RPE and PRD were recorded at 5,10,15 and 20 minutes.

13. Immediately on completion of each trial the regulator, helmet, flash hood and face piece were removed.

14. Subjects then followed a standardized cool down at 2.5 m.p.h on a level treadmill for 3 minutes.

15. After the intial weigh in, subjects were prohibited from drinking until after the final blood sample was taken, they had dried off and had been weighed again in just running shorts.

B.3 Blood Lactate

1. All blood samples were taken by a registered nurse.

2. Before each trial, an appropriate site was scrubbed with a povidone-iodine swab and a 22 guage teflon catheter was inserted into a convenient forearm vein.

3. A Hi-Flo three way stopcock (Medex) was attached to the catheter and the whole device was taped to the arm or the back of the hand.

4. The protective glove for this hand was not worn and the sleeve of the bunker coat was taped firmly above the catheterized area in order to ensure that there was no contamination of the sterile area.

5. This set up allowed the nurse to remove small blood samples (approximately 1ml) while exercise continued, with no danger to the subject.

6. This was achieved by screwing a 3cc syringe onto the 3 way valve, while the subject rested their arm on the rail of the treadmill.

7. Saline was used to keep the catheter patent between samples. The saline was withdrawn from the line and discarded before the blood sample was drawn. During GXT_{21}, GXT_{40}, SUB_{21} and SUB_{40}, a small blood sample (approximately 1ml) was removed from an indwelling intravenous catheter.

8. During GXT_{21} and GXT_{40}, the first blood sample was taken at the workload that elicited V_{TN}. The second was at the workload after V_{TN} and the third was immediately at the termination of the test. The final blood sample was taken 5 minutes post test after a standardized cool down (3 minutes at 2.0 m.p.h on a level treadmill).

9. During SUB_{21} and SUB_{40} blood samples were obtained from an indwelling catheter in a dorsal forearm vein. These samples were taken prior to the warm up, at 5,10 and 15 minutes during the test and then again at the termination of the test.

10. Immediately after sampling, 200μl of blood were transferred to a preservative tube (Yellow Springs Instruments, YSI 2315). The tube was then vortexed to mix the whole blood and preservative powder.

11. The preservative tubes were stored at -20°C until lactate analysis was performed.

12. This process was accomplished within three weeks of the sample being taken. All samples were measured in duplicate with a YSI Model 27 analyzer.

13. Calibration of the analyzer was verified with known lactate standard between each set of duplicates.

B.4 Hemoglobin [Hb] and Hematocrit (Hct)

1. For SUB_{21} and SUB_{40}, pre and postest Hct and [Hb] were measured in duplicate using the pre and 5 minute post samples taken for the blood lactate measure.
2. [Hb] was measured by pipetting 20μl of the whole blood sample into 5ml of Drabkins solution.
3. This solution was mixed well and a 1ml sample of the resulting cyanmethemoglobin was then transferred to a well polished cuvette and analysed using a spectrometer set at a wavelength of 540nm.
4. Hct was also measured at the same time as [Hb]. The remainder of the samples used for measuring blood lactate and hemoglobin were again vortexed well and 50μl was transferred to two heparinized capillary tubes.
5. The tubes were sealed at one end using Critoseal and spun in a microhematocrit centrifuge for 5 minutes.
6. The measurement of Hct was then calculated as the ratio of packed red blood cells to plasma.

APPENDIX C

PLATES

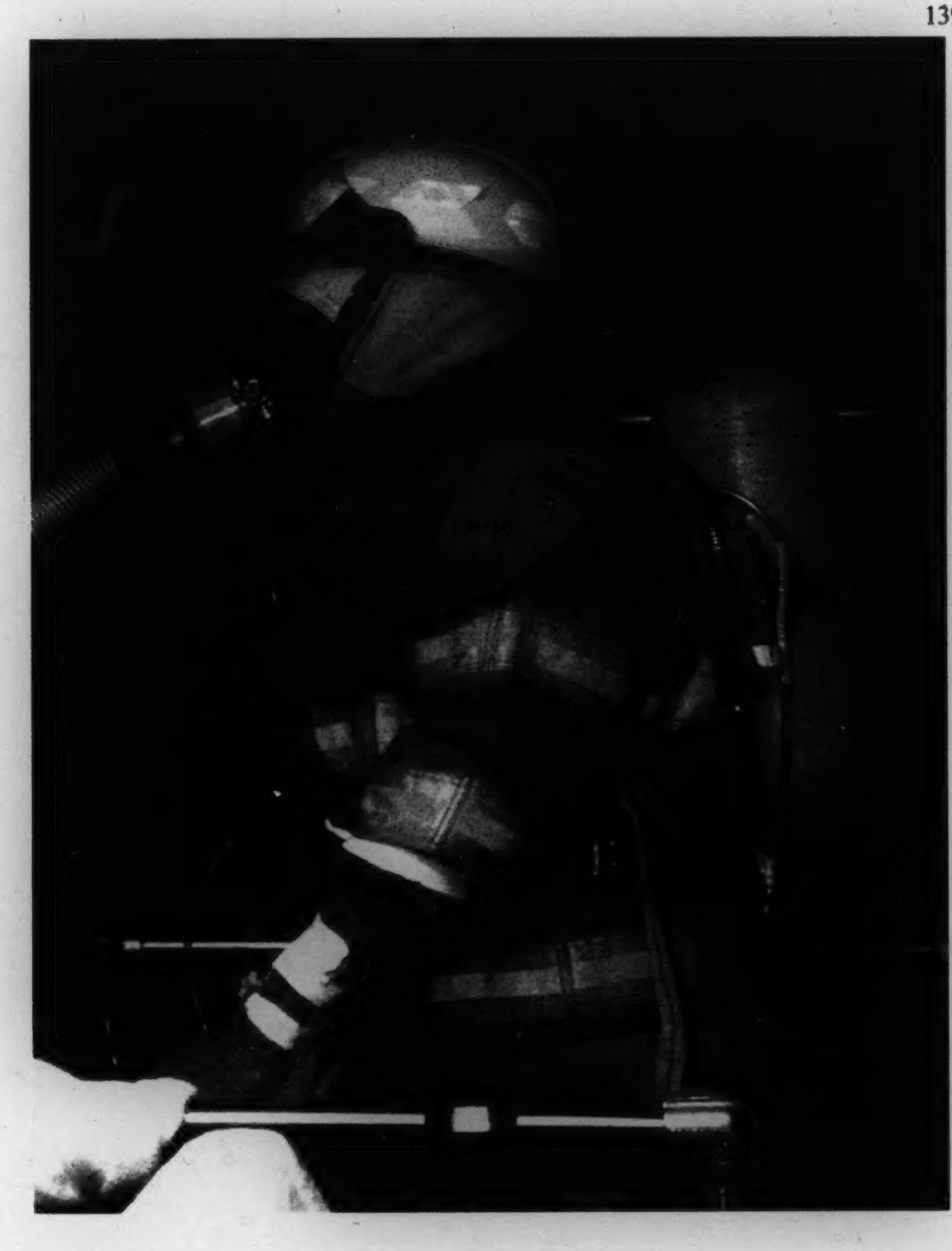


Figure C-1 : Method 1: Delivery of NOX and HOX gas mixtures through a SCOTT 4.5 gas cylinder carried by the subjects.

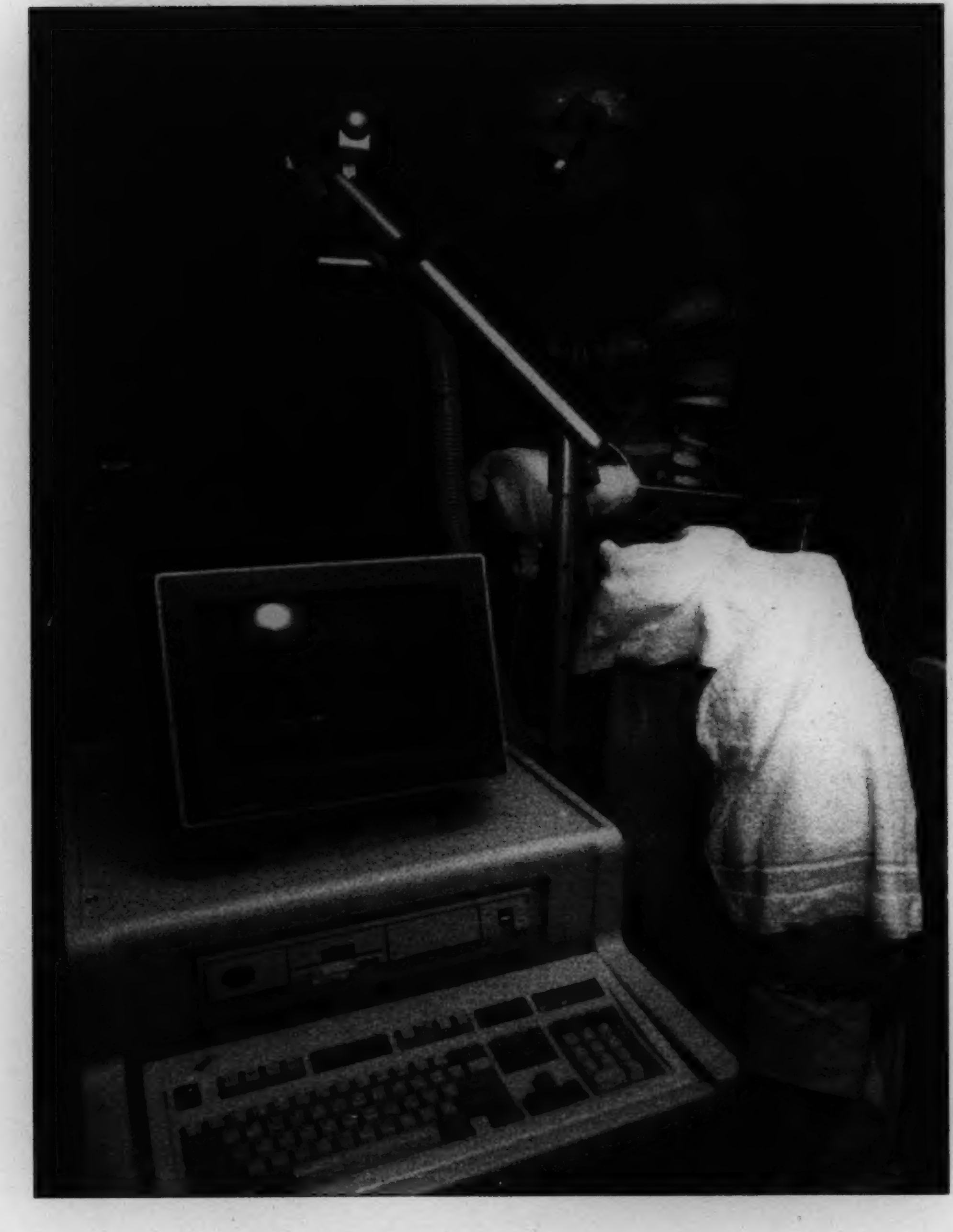


Figure C-2 : Method 2: Delivery of NOX and HOX gas mixtures from covered K-size cylinders standing adjacent to the treadmill.

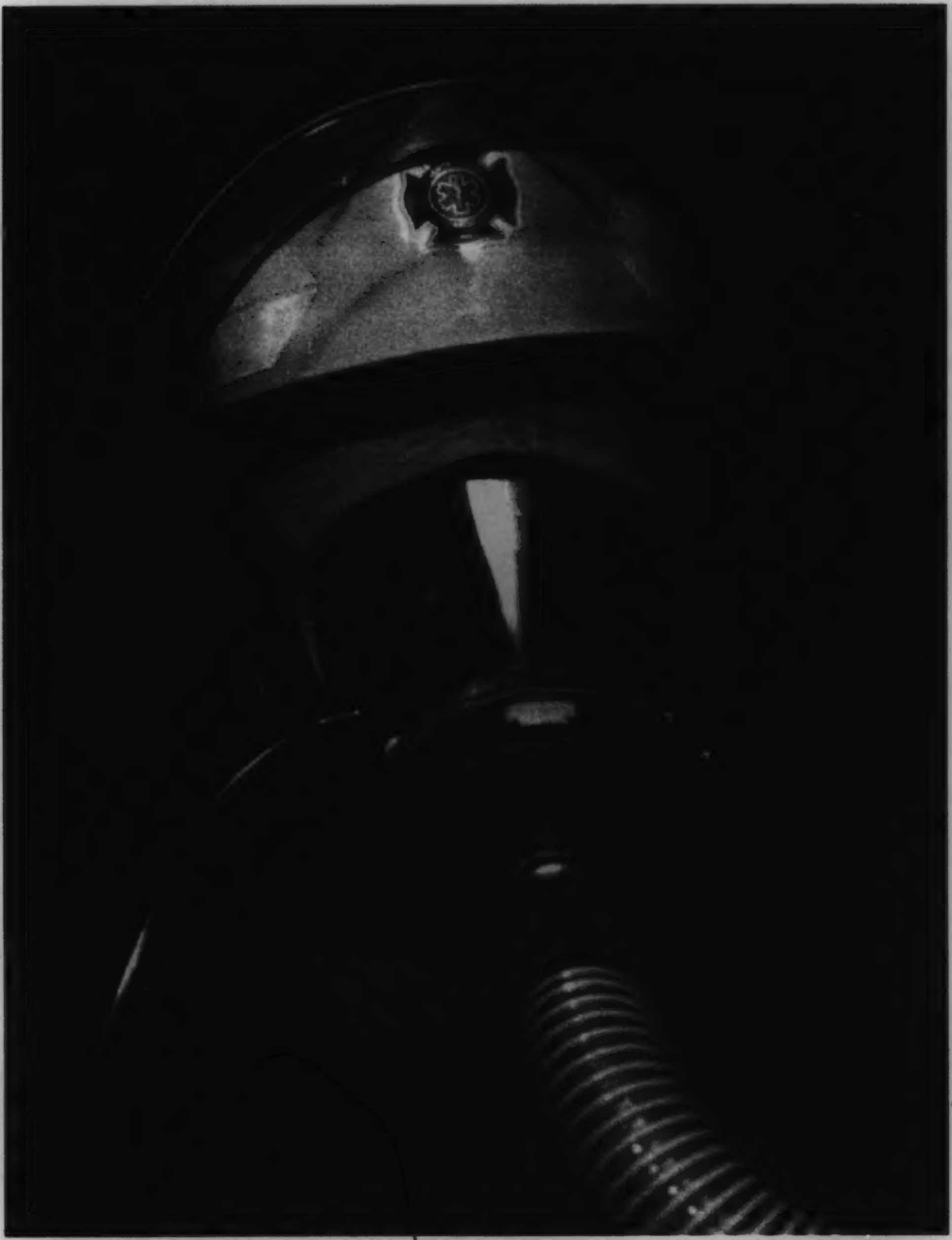


Figure C-3 : The modified mask and the specially designed plastic cone and regulator used for measuring mask pressure and expired gases, respectively.

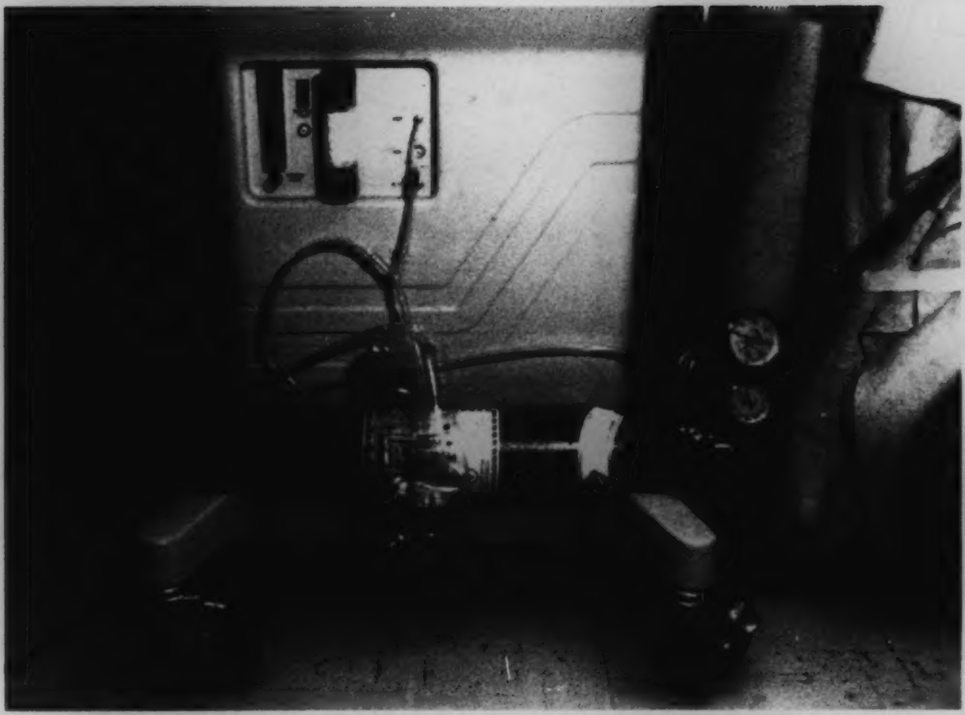
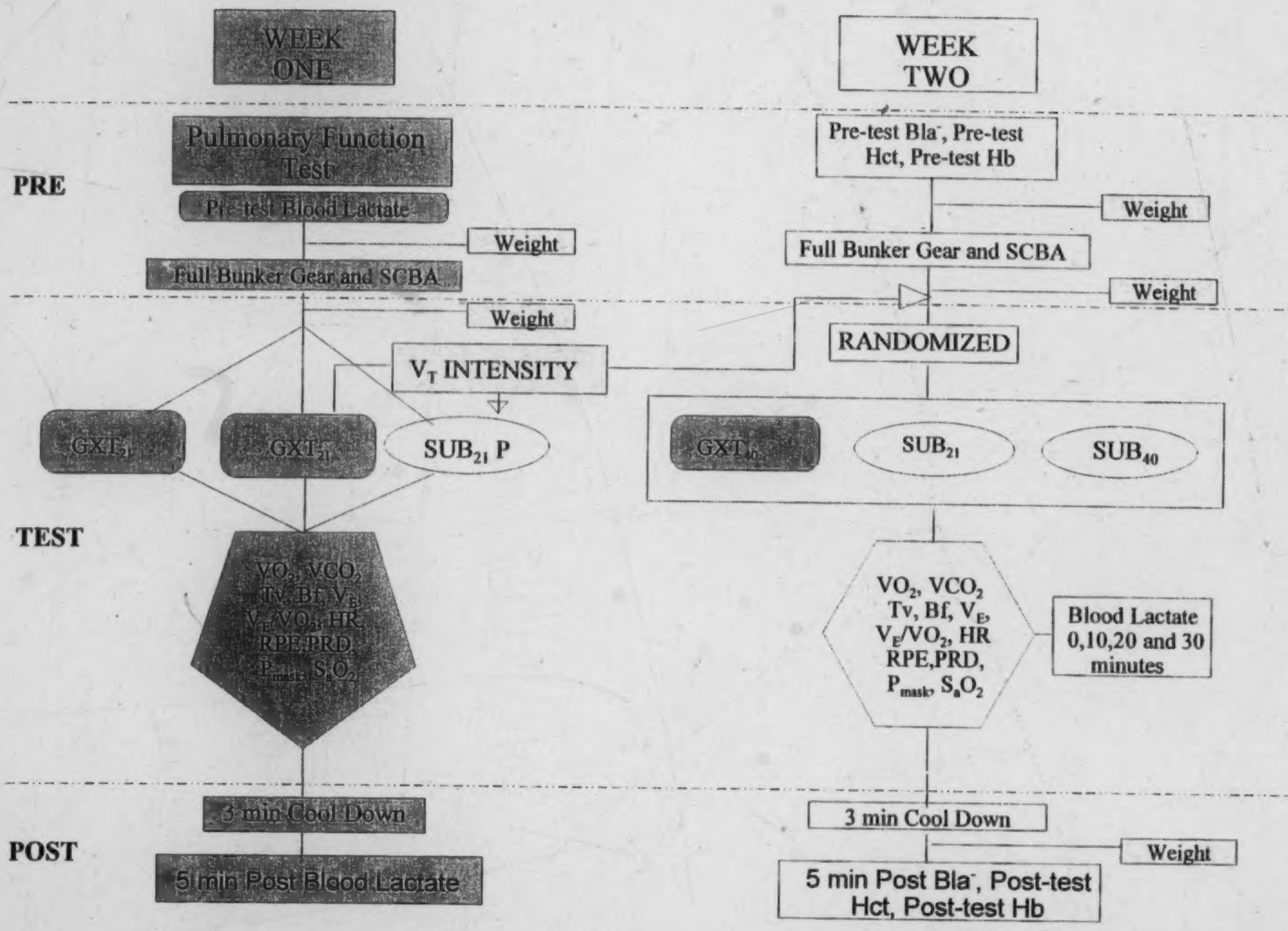


Figure C-4 : The apparatus used for measuring F_IO_2 with the 2900z MMC before all hyperoxic tests.

APPENDIX D

METHODOLOGY SCHEMATIC


WEEK ONE
WEEK TWO
PRE
Pulmonary Function Test
Pre-test Blood Lactate
Weight
Full Bunker Gear and SCBA
Weight
Pre-test Bla⁻, Pre-test Hct, Pre-test Hb
Weight
Full Bunker Gear and SCBA
Weight
RANDOMIZED
V_T INTENSITY
TEST
GXT_21
GXT_21
SUB_21 P
GXT_40
SUB_21
SUB_40
VO_2, VCO_2 Tv, Bf, V_E, V_E/VO_2, HR, RPE,PRD, P_mask, S_aO_2
VO_2, VCO_2 Tv, Bf, V_E, V_E/VO_2, HR RPE,PRD, P_mask, S_aO_2
Blood Lactate 0,10,20 and 30 minutes
POST
3 min Cool Down
5 min Post Blood Lactate
3 min Cool Down
Weight
5 min Post Bla⁻, Post-test Hct, Post-test Hb

APPENDIX E

CALIBRATION PROCEDURES

E.1 Calibration of the SensorMedics 2900z MMC for use with 40% O_2

The Calibration of the MMC was a relatively uncomplicated process and as mentioned in Chapter 5, it appears that we were relatively successful with the measurement of metabolic parameters. However, when first trying to overcome this problem, it was discovered that the software for the 2900z MMC was not compatible with an upper limit change in span gas 1 to 40%, as was initially intended. After numerous conversations with the technical staff from SensorMedics, it was ascertained that the regression calculation done by the MMC could be accurately extrapolated and used accurately with F_IO_2 concentrations as high as 80%. This assumption was validated as best as possible on our 2900z MMC, by passing a number of different known O_2 concentrations (of ≈40%) through the calibrated O_2 analyzers. Therefore, the standard 2900z mixing chamber analyzer calibration program was used to calibrated the O_2 and CO_2 sensors. For generating an accurate regression calculation that could be used at 40%, the MMC samples three different concentrations of O_2. A flush (20.93% O_2), span 1 (26.36% O_2) and span 2 (15.64% O_2). This calibration procedure was repeated at least twice for each test.

When an accurate calibration procedure was established, the only remaining hurdle to overcome, was allowing the MMC to sample an accurate F_IO_2. The 2900z MMC samples the F_IO_2 in the 20s immediately prior to the start of a test. This is achieved by drawing room air through a small piece of tubing on the anterior face of the cart. In order to allow the MMC to sample an accurate F_IO_2, a 40% O_2 calibration tank was attached to this sampling line. This was achieved using a length of 5mm inside diameter tubing, attached to the tank regulator at one end and a 1 liter reservoir bag with a flushing port at the other. Another 5 mm diameter piece of tubing then ran from the reservoir bag and attached to the sample line of the 2900z (Figure C-4). As the subject warmed up, the calibration tank, which stood behind the subject, was turned on and the gas mixture was bled through the system for approximately 60s. This flushed out all the room air from within the reservoir bag and allowed us to avoid any contamination during sampling. Therefore, immediately prior to the test the MMC sampled an accurate F_IO_2 from the reservoir bag. To ensure the accuracy of our measurements for F_IO_2 in both NOX and HOX the O_2 concentration in every tank used in this study, was checked by passing it through our analyzers at the start of each day and comparing the measured value with the certified O_2 concentration.

At the end of each test the calibration of the MMC was verified, using the standard mixing chamber analyzer calibration verify program. Results were corrected if the post calibration measurement was out by more than 0.03 from span gas values. This was felt to be more stringent than the 0.05 usually excepted for exercise testing with the 2900z during room air breathing. Likewise, it is recognized that any error found in the measurement of F_IO_2 could have a dramatic effect on the measurement of $\dot{V}O_2$. So all errors in our measurements were corrected using the 'Edit program' available on the 2900z.

APPENDIX F

INFORMED CONSENT FORM

UNIVERSITY OF ALBERTA
FACULTY OF PHYSICAL EDUCATION AND RECREATION

INFORMED CONSENT FOR THE RESEARCH PROJECT

THE EFFECT OF HYPEROXIA ON THE WORK CAPACITY OF FIREFIGHTERS

The atmosphere that we breathe contains approximately 21% oxygen, and an increase in the inspired oxygen concentration is known as hyperoxia. Hyperoxia has been shown to be a beneficial aid to exercise performance. Despite the well documented benefits, there has been relatively little research that has attempted to apply this knowledge into the very real situation of firefighting, where any increase in performance could be the difference between losing and saving lives. The aim of this study is to investigate whether the quantity of work that can be accomplished by firefighters, at an intensity similar to that required during job related tasks will vary depending on the fraction of oxygen inspired.

Participation in this project will require the completion of six tests. Three will be graded exercise tests (GXT) to exhaustion on a treadmill to evaluate anaerobic threshold and maximal oxygen uptake (V_T and VO_{2max}) while breathing either normoxic air (NOX:21% oxygen) or a hyperoxic gas mixture (HOX:40% oxygen). The second set of tests will consist of two constant load tests of 20 minutes of treadmill walking while breathing either NOX or HOX. Before completing the trials, subjects will complete a practice session to ensure that they are completely familiar with the protocol.

Throughout the data collection sessions, subjects will wear full National Fire Protection (NFPA) Standard 1500 compliant protective clothing as follows: pants; coat; helmet; gloves and Scott 4.5 Self contained breathing apparatus (SCBA) including face piece. Subjects will wear their own running shoes rather than firefighting boots in the interests of comfort and safety on the treadmill. It is expected that all six lab sessions can be completed over approximately 2 weeks. Most of the tests will be scheduled in a random order.

Each subject will be provided with a complete set of personal results as soon as possible after the study is completed. All test procedures will be fully explained and subjects will always have the opportunity to question the exact procedures that will be followed. Subjects may ask questions **at any time** during the study by contacting either:

Dr. Stu Petersen
Associate Professor
Faculty of Physical Education and Recreation
University of Alberta
Office Telephone : 492-1026
Laboratory Telephone : 492-7394

Neil Eves
Master of Science Student
University of Alberta
Home Telephone : 432-2174
Laboratory Telephone : 492-7394

The physiological tests involved in the project are explained briefly below:

Exercise Electrocardiograph (ECG) Screening

Prior to entering the experimental protocol, subjects may be requested to complete a physician supervised graded exercise test with a 12-lead ECG. This test will be conducted at the Links Clinic under the supervision of Dr. Ron Dlin. Subjects will walk at a brisk pace on a treadmill while the grade is progressively increased each minute until the point of maximal effort is reached. Blood pressure and heart rhythm will be monitored continuously to assess tolerance for heavy exercise. Including preparation, warm-up, testing and cool-down this procedure will require approximately 60 minutes.

Treadmill Test for ventilatory Threshold (V_T) and Maximal Oxygen Uptake (VO_{2max})

The ventilatory threshold is an estimation of the highest level of exercise that can be maintained for prolonged periods of time and is an excellent measure of endurance capacity. The maximal oxygen uptake is a measurement of the highest rate of oxygen utilization to produce energy during heavy exercise.

Both of these parameters will be assessed during a single graded exercise test to exhaustion on the motor-driven treadmill. Subjects will walk on the treadmill at a brisk pace and the incline will be systematically increased to increase the workload. During the test, which will last approximately 15 minutes, expired air will be collected using a permanently mounted cone which is fixed over the exhalation ports of the SCBA regulator. This air will then be analyzed by an automated Metabolic Measurement System (MMC). Breathing resistance will be calculated from measurements of pressure inside the face-piece. A pulse oximeter will be attached to a finger for measurements of oxygen saturation levels in the blood. Heart rate will be continuously measured throughout work and recovery using a telemetry system (Polar Sport-tester). This system consists of a small elastic strap worn around the chest next to the skin and a recording devise similar to a wrist watch which will be attached to the treadmill so the researchers can record results. During the test about 4 or 5 small samples of blood (approximately 1 ml each) will be drawn from a forearm vein for determination of lactate concentration. A nurse with special IV training will perform the blood collection.

Ratings of perceived exertion (RPE) will be recorded at the end of each stage of the GXT. Subjects will be familiarized with a numerical scale during the practice sessions, on which the numbers relate to the level of physical effort perceived by the subject. The numbers range from 6 to 20 with odd numbers giving descriptions from very, very light (RPE=7) to very, very hard (RPE=19). At pre-determined points during the tests, subjects will be asked to indicate a numerical rating more closely matching how they feel "overall" for the exercise that they are performing.

In conjunction with the RPE scale, Perceptions of Respiratory Distress (PRD) will also be measured using a 7 point scale, where numbers range from 0 to 6 with odd numbers giving descriptions from "My breathing is okay right now" (PRD=1) to " I can't breathe"

(PRD=7). This scale will be used at the same time as the RPE scale and subjects will be asked to indicate the rating closely matching how difficult they perceive the effort of breathing to be.

Including preparation, warm-up, the test and recovery, approximately 60 minutes will be required for this procedure.

Practice session

Prior to the two experimental trials, subjects will complete a practice session for familiarization with the exercise protocols. During this time, subjects will receive full instruction on the protocol involved with the trials and will practice the procedure. Subjects will be fitted with the full bunker gear and SCBA, and will then walk for 20 minutes at the calculated intensity for V_T, breathing a normoxic gas mixture from the SCBA. Subjects will be connected to the MMC, and the metabolic parameters will be recorded.

Including preparation, warm-up, the test and recovery, approximately 60 minutes will be required for this procedure.

Experimental Trials

Two experimental trials will be completed in random order. One of the trials will be undertaken with a normal room air concentration of oxygen (21%) and one with 40% oxygen. The trial will involve wearing the full bunker gear and the SCBA and the subjects will be connected to the metabolic measurements system. After an adequate warm-up, the subject will then connect the regulator of the SCBA and proceed to walk on the treadmill at the intensity which elicited VT for a period of 20 minutes.

During the trials, subjects will wear a Polar heart rate telemetry system that will display heart rate throughout the 20 minute work period. Ratings of perceived exertion (RPE) will be recorded at five minute interval throughout the trials. Subjects will be familiarized with a numerical scale during the practice sessions, on which the numbers relate to the level of physical effort perceived by the subject. The numbers range from 6 to 20 with odd numbers giving descriptions from very, very light (RPE=7) to very, very hard (RPE=19). At pre-determined points during the tests, subjects will be asked to indicate a numerical rating more closely matching how they feel "overall" for the exercise that they are performing.

In conjunction with the RPE scale; Perceptions of Respiratory Distress (PRD) will also be measured using a 7 point scale, where numbers range from 0 to 6 with odd numbers giving descriptions from "My breathing is okay right now" (PRD=1) to " I can't breathe" (PRD=7). This scale will be used at the same time as the RPE scale and subjects will be asked to indicate the rating closely matching how difficult they perceive the effort of breathing to be.

During the experimental trials blood samples will be taken every five minutes. The subjects will have a venous catheter inserted by a registered nurse into a vein on the forearm near the wrist. The protective glove for this hand will not be worn which will allow the nurse to remove a small blood sample, at the pre-determined times. The last blood sample will be drawn after exactly 5 minutes standard active recovery. This data will allow evaluation of the physiological responses to each exercise protocol.

Including preparation, warm-up, the test and recovery, approximately 60 minutes will be required for this procedure

Pulmonary Function Tests

At a convenient time prior to one of the tests listed above, resting pulmonary function measurements will be taken using a computerized spirometer system. These measures will include static lung volumes, flow rates, and maximal ventilatory volume. Normally about 15 minutes is required for these procedures, which includes orientation, practice and actual testing.

Time Commitment

In order to complete all experimental procedures, subjects will need to visit the lab on 6 separate days. It is estimated that the total time required for participation in the study should not exceed 8 hours, over a period of approximately 2 weeks. All tests will be scheduled at mutually convenient times.

Subjects must agree to participate to the best of their ability and must be rested, adequately fed and hydrated at the start of each session. It is important to note that consistency in time of day and pretest behavior (including exercise, rest and nutrition) is a critical aspect of compliance as a subject. Subjects must agree to consistently control these factors in consultation with either Dr. Petersen or Neil Eves.

Risks

Subjects should understand that the tests require a strenuous to maximal physical and metal effort. **However, both the physical and mental stresses involved in these tests should be no greater than might normally be experienced during firefighting operations or normal training and competition for most sports.** While there is virtually no serious health risk to healthy, physically active adults undertaking the type of exercise challenges utilized in this study, transient discomfort, heat stress, dizziness, and/or nausea may occur.

The difficulty of the exercise tests is increased by wearing the bunker gear and SCBA. It is especially important for subjects to be careful while walking on the treadmill in firefighting gear to avoid stumbling. Extra care will be taken by the lab staff to ensure safety in this procedure.

The blood sampling procedures are invasive (the skin is punctured) and with any such problems there is a risk of infection. The risk of infection is virtually eliminated when rigorous procedures regarding sterility of instruments are followed. A risk of bruising or hematoma at the puncture site exists if sufficient pressure is not applied when the needle is withdrawn. Again, the risk is minimal if the subject follows the guidelines provided by the registered nurse present. All lab procedures involving collection and handling of fluids are conducted under the strict guidelines of Occupational Health and Safety (OHS).

The tests will be administered by qualified personnel under the supervision of Dr. Petersen. Subjects should understand that the laboratory staff conducting the tests may terminate any procedures if abnormal responses become evident. Subjects should also acknowledge their responsibility to inform the laboratory personnel of any symptoms of pain, illness, or undue fatigue they may experience during the study procedures.

Confidentiality of Information

The information collected in the study will be maintained in a confidential fashion. Test results and personal data will be assigned a code designation so that personal identification will not be possible. The data will be stored in a secure location accessible only to Dr. Petersen, Neil Eves and any research assistants involved in the project. Following the completion of the study the coded results will be released to each individual subject only. Any research publications as a result of the study will not identify subjects by name.

Consent

I have read this document and fully understood the procedures and risks associated with the study. I agree to participate in this project with the understanding that I may withdraw my participation at any time, for any reason, without penalty or consequence. I will receive a copy of this signed document and after the study is complete I will receive a full report of my results.

Signatures

Subject Name (please print)____________________________________

Subject______________________________ Date__________________

Contact Telephone____________________________

Investigator___________________________ Date__________________

APPENDIX G

REVISED PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (rPAR-Q)

PAR-Q
REVISED PHYSICAL ACTIVITY READINESS QUESTIONNAIRE

1. Has your doctor ever said that you have a heart condition and recommended only medically approved physical activity?
   Yes______ No______
2. Do you have chest pain brought on by physical activity?
   Yes______ No______
3. Have you developed chest pain at rest in the past month?
   Yes______ No______
4. Do you lose consciousness or lose your balance as a result of dizziness?
   Yes______ No______
5. Do you have a done or joint problem that could be aggravated by the proposed physical activity?
   Yes______ No______
6. Is your doctor currently prescribing medication for a blood pressure or heart condition (e.g., diuretics or water pills)?
   Yes______ No______
7. Are you aware, through your own experience or a doctor's advice, of any reason against your exercising without medical approval?
   Yes______ No______

Note.

1. This questionnaire applies to those 15-69 years of age.
2. If you have a temporary illness, such as a fever, or are not feeling well at this time, you may wish to postpone the proposed physical activity.
3. If you are pregnant, you are advised to discuss the "PAR-X for Pregnancy" form with your physician before exercising.
4. If there are any changes in your status relative to the above questions, please bring this information to the attention of your fitness professional.

I have read, understood, and completed this questionnaire.

Signature______________________________ Date__________

Signature of Parent or Guardian (if under 18)__________________

Witness______________________________ Date__________

APPENDIX H

ETHICS APPROVAL

Faculty of Physical Education and Recreation
University of Alberta

Proposal No. 98-0219-01

Ethics Review Approval

The Ethics Committee of the Faculty of Physical Education and Recreation (University of Alberta):

Name	Position
Dr. Jane Watkinson	Professor and Associate Dean
Dr. Romeo Chua	Assistant Professor
Dr. Dick Jones	Professor (Pulmonary Medicine)

have reviewed the proposal entitled:
The effect of hyperoxia on the work capacity of firefighters

submitted by
S.R. Petersen and N.D. Eves

✗ finds it within acceptable standards for human experimentation

finds it within acceptable standards subject to the following revisions:

finds it unacceptable in its present form

Jane Watkinson

Dr. Jane Watkinson, Chair
Faculty Ethics Committee

March 12, 1998
Date